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Margvelashvili, Ann ; Zollikofer, Christoph P E ; Lordkipanidze, David ; Tafforeau, Paul ; Ponce de León, Marcia S

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Authors: Ann Margvelashvili[a,b]*, Christoph P.E. Zollikofer[a], David Lordkipanidze[b], Paul Tafforeau[c], Marcia S. Ponce de León[a]

Institutions: [a] Anthropological Institute and Museum, University of Zurich, Winterthurerstrasse 190, CH-8057, Zurich, Switzerland.

[b]Georgian National Museum, Purtseladze 3, 0105, Tbilisi, Georgia.

[c] European Synchrotron Radiation Facility, 6 Rue Jules Horowitz, 38043 Grenoble Cedex, France.

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Key words: aging, tooth wear, caries, periodontitis

***Corresponding author**: Ann Margvelashvili, Anthropological Institute and Museum, University of Zurich, Winterthurerstrasse 190, CH-8057, Zurich, Switzerland; Tel: 0041 446355441; Fax: 0041 446356804; e-mail: margvelashvili@aim.uzh.ch or animargvel@gmail.com

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Comparative analysis of dentognathic pathologies in the Dmanisi mandibles

Objectives: Due to the scarcity of the fossil record, *in-vivo* changes in the dentognathic system of early *Homo* are typically documented at the level of individual fossil specimens, and it remains difficult to draw population-level inferences about dietary habits, diet-related activities and lifestyle from individual patterns of dentognathic alterations. The Plio-Pleistocene hominin sample from Dmanisi (Georgia), dated to 1.77 million years ago, offers a unique opportunity to study *in-vivo* changes in the dentognathic system of individuals belonging to a single paleodeme of early *Homo*. **Materials and Methods:** We analyze dentognathic pathologies in the Dmanisi sample, and in comparative samples of modern Australian and Greenlander hunter-gatherer populations, applying clinical protocols of dentognathic diagnostics. **Results:** The Dmanisi hominins exhibit a similarly wide diversity and similar incidence of dentognathic pathologies as the modern human hunter-gatherer population samples investigated here. Dmanisi differs from the modern population samples in several respects: At young age tooth wear is already advanced, and pathologies are more prevalent. At old age, hypercementosis is substantial. **Discussion:** Results indicate that dentognathic pathologies and disease trajectories are largely similar in early *Homo* and modern humans, but that the disease load was higher in early *Homo*, probably as an effect of higher overall stress on the dentognathic system.

Key words: aging, tooth wear, caries, periodontitis

The Plio-Pleistocene site of Dmanisi has yielded a large sample of hominin fossils associated with abundant and diverse faunal remains and Mode I stone artifacts (Gabunia et al. 2001; Ferring et al. 2011; Mgeladze et al. 2011). Occupation of the site began shortly after 1.85 Ma. The fossil hominin finds are dated to 1.77 Ma (Ferring et al. 2011). Dmanisi thus represents the earliest evidence of *Homo* outside of Africa. The well-preserved cranial, mandibular and postcranial remains of five individuals are unique in offering detailed insights into patterns of morphological variation within a paleodeme of early *Homo*, and into patterns of growth and aging (Lordkipanidze et al. 2013; Margvelashvili et al. 2013; Zollikofer et al. 2014). The cranio-mandibular morphology of the Dmanisi paleodeme has been described previously (Gabunia and Vekua 1995; Gabunia et al. 2000; Gabunia et al. 2001; Gabounia et al. 2002; Lordkipanidze et al. 2005; Lordkipanidze et al. 2006; Rightmire et al. 2006; Rightmire and Lordkipanidze 2010; Lordkipanidze et al. 2013; Margvelashvili et al. 2013). Cranial and mandibular morphological variation within the Dmanisi paleodeme is wide, but well within the range of variation seen in modern humans populations, and in populations of chimpanzee subspecies (Skinner et al. 2006; Lordkipanidze et al. 2013; Margvelashvili et al. 2013; Zollikofer et al. 2014). Variation in size, shape and dentognathic features among the Dmanisi mandibles has received special attention and has been studied to address questions of phylogeny, taxonomy, sexual dimorphism, diet, aging, and *in-vivo* modification of the dentognathic system (Gabounia et al. 2002; Skinner et al. 2006;

Margvelashvili et al. 2013; Martín-Francés et al. 2013; Bermudez de Castro et al. 2014; Schwartz et al. 2014; Zollikofer et al. 2014).

Wear-related dentognathic remodeling has been shown to be a major factor underlying mandibular variation in modern human hunter-gatherer (HG) populations (Begg 1954; Kaifu et al. 2003), and in the Dmanisi sample (Margvelashvili et al. 2013). Wear-induced changes in dental morphology trigger compensational remodeling mechanisms of the dentognathic system, such as continuous dental eruption, mesial drift of the posterior teeth, and lingual tipping of the anterior teeth. Overall, these mechanisms contribute substantially to within-populations dentognathic variation.

While wear-related dentognathic changes in the Dmanisi mandibles are now well documented, a comprehensive analysis of dentognathic pathologies (DP) is still missing. The first aim of this study is to provide the relevant empirical data for all the Dmanisi mandibles. The second aim is to analyze and interpret this body of evidence in a well-defined comparative framework of DP in modern human HG populations.

Establishing a comparative framework is important because still relatively little is known about patterns and prevalence of DP in early *Homo*. The adolescent *H. erectus ergaster* specimen KNM-WT 15000 (1.5 MA) exhibits tooth agenesis of both mandibular M3s, and linear enamel hypoplasias (Dean and Smith 2009). The specimens from Sima del Elefante (estimated to 1.3 MA) and Sima de los Huesos (0.3 MA) display a wide diversity of pathologies such as alveolar lytic lesions, hypercementosis and dental calculus (Martinon-Torres et al. 2011), as well as periodontal disease, periapical infections, and even osteitis (Gracia-Téllez et al. 2013). The Broken Hill maxillary dentition (0.3-0.125 MA) displays hypercementosis, multiple lesions of cariogenic origin, periodontal disease, and possible effects of hyposalivation (Koritzer and St Hoyme 1979; Puech et al. 1980; Bartsiokas and Day 1993; Lacy 2014b).

In mid-to-late Pleistocene hominins such as the Neanderthals, dental pathologies are comparatively well documented (Smith 1976; Trinkaus 1978; Trinkaus 1985; Frayer and Russell 1987; Lalueza et al. 1993; Tillier et al. 1995; Skinner 1996; Trinkaus et al. 2000; Lebel and Trinkaus 2002; Guatelli-Steinberg et al. 2004; Walker et al. 2011; Topić et al. 2012; Lozano et al. 2013; Lacy 2014a). A wide range of pathological features has been reported, from developmental defects such as linear enamel hypoplasia, to *antemortem* tooth loss, and *in-vivo* effects such as interproximal grooves reflecting the habitual use of toothpicks. Compared with Neanderthals, little information is available on DP for early *Homo sapiens.* Recently a case of malocclusion (cross-bite) has been reported in the Qafzeh 9 individual (dated to 94-115 ka), with additional midline deviation and incisor rotations (Sarig et al. 2013). Caries has been reported in Skhul 2, and in Qafzeh specimens 3, H4, 4, 7, 9, and 11 (Tillier et al. 1995; Trinkaus and Pinilla 2009; Lacy 2014a).

Recently, Lacy (2014a) used a population-level approach to analyze oral health conditions in a large sample of Late Pleistocene Eurasian hominins. While the results of Lacy's PhD thesis have not yet been published in detail, it is worth mentioning that studying dentognathic data in terms of broad taxonomic, geographic and temporal groups reveals significant large-scale patterns. Most importantly, it appears that the oral health conditions of the Neanderthals were compromised compared to Early Upper Paleolithic modern humans, but that during the Last Glacial Maximum the oral health status of the latter group declined as an effect of harsher environmental conditions (Lacy 2014a).

Due to the scarcity of the fossil record of early *Homo*, most DP studies follow an individual-centered, descriptive approach that is characteristic of paleopathological case studies. These early *Homo* case studies often lack a well-defined quantitative comparative context and do not account for the potential significance of within-population variation. They thus tend to draw far-reaching conclusions from single specimens regarding taxon-specific masticatory function, dietary habits, and the paramasticatory use of the dentition (Martinon-Torres et al. 2011; Gracia-Téllez et al. 2013; Martín-Francés et al. 2013; Bermudez de Castro et al. 2014). Overall, thus, the results of paleopathological dentognathic studies in early *Homo* are difficult to integrate into a wider comparative framework, and specific hypotheses regarding causes and mechanisms of pathogenesis cannot be tested explicitly.

To address these issues, we further develop the population-level perspective proposed earlier (Margvelashvili et al., 2013; Lordkipanidze et al. 2013; Lacy 2014a). Specifically, we propose the following quantitative approach for the comparative analysis of dentognathic pathologies:

- Fossil hominin dentognathic pathologies are classified and documented according to clinical standard definitions, such that they can be compared with pathologies in modern human pre- or post-industrial populations.
- A scoring scheme is implemented for the quantitative assessment of the location and grade of pathologies in individual fossil specimens. Equivalent procedures are used to analyze comparative samples from modern HG populations.
- Data are integrated at the level of populations. This permits analysis of patterns of within- and between-population variation in dentognathic pathologies. Modern HG populations representing different lifestyles are used as a frame of reference to draw inferences on developmental, behavioral, and environmental factors underlying dentognathic pathologies in fossil hominin populations.

The Dmanisi mandibles offer a unique opportunity to implement this approach because they display a wide range of variation in individual age, dental wear stage, and DP. The comparative sample used here comprises Australian aboriginal and Greenlander HG populations, which also exhibit a wide range of variation in age, wear and pathological features, and for which dietary

patterns, masticatory and non-masticatory tooth use, and environmental conditions are well documented.

The goal of these comparative analyses is to establish links between patterns and processes of DP in modern populations, and use this information to infer process from pattern in fossil populations. As shown in Figure 1, the cause-effect network of DP is complex, but it is possible to discriminate between four main factors (processes) causing dentognathic alterations and pathologies.

1) Genetic and developmental factors. Genetic and developmental disorders, which cause malformation of dentognathic elements.

2) Dental wear. Dental mesowear has been shown to be a major factor underlying *in-vivo* remodeling of the dentognathic system (Ungar et al. 1997; Kaifu 1999; Kaifu et al. 2003; d'Incau et al. 2012; Margvelashvili et al. 2013). Mesowear is also known to be the major long-term cause of DP (Smith 1976; Richards and Brown 1981). Mesowear itself is typically caused by a combination of mechanisms such as attrition (wear from tooth-to-tooth contact), abrasion (wear from tooth-to-object contact), erosion (dental crown destruction by chemical processes) and abfraction (wedge-shaped defects at the cemento-enamel junction) (Habsha 1999; Lucas et al. 2013). Also, rates of dental wear depend on the type of diet and on non-dietary factors such as paramasticatory activities and environmental conditions (Lucas et al. 2013). Because the relative contribution of these factors and mechanisms remain unknown, dental wear scores cannot be used as proxies for individual age in cross-population studies. However, within a given population (i.e., given largely similar dietary, behavioral and environmental conditions), dental wear tends to be correlated with individual age (Miles 1962; Lovejoy 1985; Miles 2001).

3) Trauma and behavioral patterns. Damage to the dentognathic system due to traumatic events or culturally mediated behaviors, typically resulting in partial or complete loss of teeth, and/or bone fractures/lesions.

4) Infections. Generalized and local dental and bony infections and periodontal diseases.

The complex cause-effect network of DPs (Fig. 1) implies that a given DP pattern can be brought about by various combinations of underlying factors. Equifinality thus limits inferences of DP process from DP pattern. We thus focus on the comparison of population-specific patterns of DP, while inferences regarding causes and processes (such as diet composition and dietary habits) must remain tentative. Using the proposed comparative approach, we test the following hypotheses:

Hypothesis 01: The diversity (different types) of dentognathic pathologies is similar in Dmanisi and the modern HG population samples.

Hypothesis 02: The prevalence (frequency) of dentognathic pathologies is similar in Dmanisi and the modern HG population samples.

Hypothesis 03: The temporal (developmental and age-related) patterning of dentognathic pathologies is similar in Dmanisi and the modern HG population samples.

The modern human comparative sample used here represents two HG populations, Australians and Greenlanders, who are known to exhibit substantial differences in dietary niches, food processing habits, and masticatory/paramasticatory habits (see Materials). These differences are expected to influence the population-specific diversity, prevalence and spatiotemporal patterning of DP. Using these populations as a comparative sample thus guarantees the widest-possible spectrum of modern DP patterns. The above hypotheses are tested by assessing three types of contrasts: (1) contrasts between early Pleistocene and modern HGs: Dmanisi versus modern human HGs (comprising both populations); (2) contrasts between modern human HGs: Australians versus Greenlanders; (3) contrasts between Pleistocene and modern populations: Dmanisi versus each of the modern human HG populations. If any of the hypotheses H01 – H03 is falsified, the alternative hypothesis is examined to assess the role of factors 1 – 4 bringing about the observed differences between groups. Hypothesis falsification criteria are as follows:

H01 is rejected if the Dmanisi sample exhibits DPs that are not present in the modern human comparative sample.

H02 is rejected if the prevalence of pathologies in the Dmanisi sample is statistically different from the prevalence in the modern comparative samples.

H03 is rejected if the incidence of pathologies (prevalence as a function of dental wear) in the Dmanisi sample is statistically different from that in the modern comparative samples.

MATERIALS AND METHODS

Sample (see Table 1)

The Dmanisi sample comprises mandibles D2735, D211, D2600, D3900, and several isolated teeth belonging to mandible D2735: the right (D2678) and left (D2723) canines, right (D2854) and left (D3698) second incisors, and a recently discovered right third molar (D5099).

The comparative HG sample consists of Australian aborigines (*N*=26) from the Duckworth Collection (Leverhulme Centre for Human Evolutionary Studies) representing different populations, and Greenlanders (*N*=49) from the Collections of the Anthropological Institute and Museum of the University of Zurich and the Greenland Museum of the University of Copenhagen. Both modern human HG population samples are heterogeneous in the sense that they comprise specimens from various localities (see Table 1). The width of the ecocultural

niche represented by each sample thus tends to be wider than that of local HG groups, with the effect that DP diversity in the samples used here is potentially larger than in single groups.

The following observations characterize commonalities and differences between Australian and Greenlander ecocultural niches: Australian aborigines are nomadic HGs living in a wide variety of ecosystems. Their diet mostly consists of animal foods, but also vegetables with high fiber content, and carbohydrate-rich "bush-food", such as fruits, seeds, leaves, tubers, bush honey etc. (O'Dea 1988; Brand-Miller and Holt 1998). Australian HGs do not store food (Lourandos 1997; reviewed in Rowley-Conwy 2001). Greenlanders are also nomadic HGs (Arnett Jensen 2003), but they subsist on a specialized diet, which mostly consists of protein-rich animal foods hunted and/or gathered locally, including both land and marine mammals (Sharma 2010). Also, Greenlanders have been reported to be exposed to high masticatory loads, and to make extensive use of their dentognathic system in paramasticatory activities (Merbs 1968; Clement et al. 2012).Food is stored to buffer seasonal fluctuations in resource availability. Both Australians and Greenlanders make extensive use of tools for food-processing.

Imaging

Microscopy. Pathologies were recorded and classified with various microscopic techniques. Occlusal microwear and mesowear in the Dmanisi hominins was examined with a Leica stereomicroscope (MZ 12.5), and further detail was documented with a Scanning Electron Microscope (SEM JEOL 6360 VP). We did not record dental microwear of the HG comparative sample. For the analysis of enamel microwear and scratching in Dmanisi hominins, molds were taken from the original specimen with the low-viscosity material Betasil Vario Intro Kit Putty (Müller-Omicron GmbH & Co.KG), and casts were produced with Epo-Tek 301 (Epoxy Technology). The SEM imaging of the cervical dental surfaces of the D2735 specimen was performed as follows: a primary mold was taken from the original specimen with the low-viscosity material Optosil-Xantopren VL plus (Heraeus Kulzer GmbH). Casts were produced with Crystal Clear (Smooth-On), coated with 16nm of platinum and examined with SEM at a variety of magnifications (23x, 35x, 60x, 85x).

Medical computed tomography (CT). Tomographic data were acquired with multislice helical CT in axial and coronal orientations (collimation 0.625 mm, pitch 0.75). Cross-sectional images (512 × 512 pixels) were reconstructed at 0.1 to 0.2 mm isotropic voxel volumes. The CT data volumes were transferred to a high-performance graphics workstation. Data analyses were performed by means of softwares VGStudioMax 2.1 (www.volumegraphics.com/) and Avizo (http://www.vsg3d.com/avizo/overview).

Synchrotron tomography. Synchrotron radiation tomography was used for in-depth evaluation of the intra-osseous bone and root modifications in mandibles D2735 and D2600. These specimens were scanned using propagation phase contrast microtomography (Tafforeau et al.

2006) on the biomedical beamline ID17 of the European Synchrotron Radiation Facility (ESRF). The beam was monochromatized using a double-Laue Silicon 111 crystal monochromator with bended crystals, and set to an energy of 96 keV. The sample-detector distance was 5 meters. The detector was composed of a 16 micron thick gadox (gadolinium oxisulfide) scintillator screen put in close contact with a parallel fiber optic taper doped with cerium, this one being in direct contact with a demagnifying taper fixed to the CCD FreLoN camera 2K14 (Labiche et al. 2007). This setup produces data with an isotropic voxel size of 45.7 microns. Each tomographic scan was covering 6.5 mm vertically. In order to optimize the data quality, the sample was moved by 3 mm vertically between each scan such that each part was scanned twice with different parts of the detector. Two double complete scans were performed this way and then later combined. Each final reconstructed slice had data from four slices acquired independently. Each sub-scan was performed using 5000 projections of 0.15 s over 360 degrees with the center of rotation on the right side of the projections in order to double the lateral field of view of the detector. The reconstruction was performed using the filtered back-projection algorithm. Two reconstruction modes were used: edge detection mode (direct reconstruction of the propagation phase contrast data) and single-distance phase retrieval mode using an adapted algorithm from Paganin (2002), coupled with a 3D unsharp filter to compensate the blurring induced by the phase retrieval (Sanchez et al. 2012). In addition to the classical flatfield/ darkfield correction, the ring artifacts were firstly attenuated by using a filtered median of the average of all the projections of each scan for each series. After reconstruction and combination of the 4 scans, the data were converted into tif file stacks with a pixel depth of 16 bits. The final residual ring artifacts were corrected on the slices using an algorithm developed at the ESRF (Lyckegaard et al. 2011).

Measurements

Dental wear was scored using an extended Molnar scale (Molnar 1971; Margvelashvili et al. 2013) (see Fig. 2 for dental wear frequency distribution per population). In order to evaluate dentoalveolar structures and structural changes due to pathology we established a dentognathic pathology classification protocol, which is based on existing clinical classifications (Le Fort 1901; Bjork A et al. 1964; Baume LJ et al. 1973; McNeill et al. 1980; Clarkson and O'Mullane 1989; Varrela and Paunio 1994; Ramachandran Nair et al. 1996; Armitage 1999; Floyd et al. 1999; Lieverse 1999; Huumonen and Ørstavik 2002; Bakland and Andreasen 2004; Nair 2004; Schulze et al. 2006; Hassan R. and Rahimmah 2007; Ismail et al. 2007; Selwitz et al. 2007; Buitrago-Téllez et al. 2008; Ogden 2008; Pinheiro et al. 2008; Rosenberg et al. 2010). We grouped DP features into 9 classes, and scored each feature according to location, and according to presence/absence or level of expression. Classes, features and scoring criteria are defined in Appendix I and summarized in Table S1.

Statistical analyses

Hypothesis H01 (spectrum of DP) was tested by direct comparisons of presence/absence data for all DP features (as specified in Appendix I and Table S1). Hypothesis H02 (prevalence of DP) was tested with resampling procedures. These procedures assess the likelihood that the pattern of DP seen in the three tooth-bearing Dmanisi mandibles (D2735, D211 and D2600) can also be observed in triplets of modern HG mandibles of comparable dental wear stages. To this end, frequency distributions for 11 DP features (3a,3b; 4a,4b; 5a-c, 6a,6b; 7a,7c; 8a, 9a; see Table S1) were calculated for each modern HG population sample, and two dental wear groups: "early wear" (Molnar stage 1-3), and "late wear" (Molnar stage 7-8). These groups were designed to match the Dmanisi sample (mandibles D2735 and D211 represent early wear, while D2600 represents late wear). The modern DP frequency distributions were then used to create (i.e., resample) "virtual" specimen triplets matching the wear-stage distribution of the three Dmanisi mandibles (two early-wear specimens plus one late-wear specimen). For each HG population, 1000 triplets were resampled, and each triplet's DP pattern was compared with the actual DP pattern of the Dmanisi triplet. As a measure of dissimilarity *D* between triplets, the "cityblock" distance was used, which was evaluated as follows: all DP scores were normalized (such that scores range between 0 and 1), between-triplet differences were evaluated for all features, and the absolute differences were summed up to obtain the dissimilarity metric *D*.

Hypothesis H03 was tested by comparing wear-related incidences (i.e. rates) of DP in Dmanisi and the modern HG samples. To this end, three DP variables [pulp chamber reduction (4b), local infections (6b), and antemortem tooth loss (8a)] were regressed against dental wear, and group-specific slopes (modern HG samples and Dmanisi) were compared. The slope evaluated for the Dmanisi sample was then compared with the frequency distribution of slopes evaluated from 80 random subsamples of the modern HG population data, where each subsample was represented by N=9 specimens (one per dental wear stage).

All analyses were performed with softwares JMP (http://www.jmp.com) and MATLAB (http://ch.mathworks.com/products/matlab/).

RESULTS

The results of this study are structured as follows: First, we provide detailed descriptions of the DP of each Dmanisi mandible. Second, overall patterns of DP are described for the two modern human populations. Third, the results of the quantitative comparative analyses of the three populations are presented.

Dmanisi dentognathic pathologies

The youngest individual of the Dmanisi paleodeme is a subadult that is represented by cranium D2700 with associated mandible **D2735** (Vekua et al. 2002; Rightmire et al. 2006), and various postcranial elements (Lordkipanidze et al. 2007). Incisors (I), canines (C) and the right third

molar (RM3) were dissociated from the mandible *postmortem*. Both I2s, Cs and RM3 were found in isolation and can be repositioned in their well-preserved sockets.

Overall tooth wear is moderate (grade 2) but the first permanent molars (M1) show substantial occlusal wear (grade 4) with dentine exposure and tertiary dentine formation in the distal portion (Margvelashvili et al. 2013). [Note that the paleoanthropology literature typically uses the term "secondary dentine formation" for both secondary and tertiary dentine formation. Primary dentine is deposited during tooth formation; secondary dentine is formed continuously, past dental eruption and into adulthood, and tertiary (or reparative) dentine is formed as a reaction to dental injury (Foster et al. 2013)]. Additionally, the specimen shows asymmetric tooth wear, with the right M1 (RM1) being slightly more worn than the left M1 (LM1). The alveolar border is inferior to the anatomical cervices of the dentition, the tip of the furcation of M1s is visible, and interdental alveolar crests are slightly rounded in the posterior dentition. Interproximal wear is generally only weakly expressed. It is most conspicuous on the approximal surfaces between M1s and P4s. On the distal surface of the RM2, a small wear facet is visible (RM3 was lost *postmortem*).

The D2735 mandible bears no external evidence of an LM3 tooth socket, and neither medical CT nor synchrotron data show evidence of a tooth bud. The retromolar area (distal to M2) on the left side is shorter than the corresponding area on the right side, resulting in asymmetrical mandibular corpus lengths with a difference of 4 mm between sides (Fig. 3). We conclude that the left M3 tooth bud has failed to form.

The left maxillary P4 of the associated D2700 cranium is compressed mesio-distally, elongated bucco-lingually and lacks a mesial marginal ridge, altogether indicating malformation. The tooth is rotated along its main axis by about 90°, such that its palatinal cusp projects mesially and is erupted palatinally in relation to the dental arcade.

Enamel hypoplasia (EH) is present on the I2s, Cs, third premolars (P3s), P4s, M2s, and RM3. The extent of the defect is less than 1/3 of the tooth crown area for I2s, P3s, P4s, LM2 and RM3, but between 1/3 and 2/3 for Cs (Fig. 4) having smooth and rounded margins. The hypoplasia of the Is is minimal, and is pit-like rather than linear. Canines show clear linear defects expressed in several bands surrounding the labial surface of the tooth, although the depth of the lesion is very superficial. P3s are the most affected ones where the developmental defect has a moderate depth on the cervical circumference of the crown. Enamel hypoplasia in P4s, M2s and RM3 EH is lightly expressed.

The labial surfaces of the I2s exhibit irregular *in-vivo* scratches covering the whole crown area (Fig. 5A). The principal orientation of the scratches is from superior-left [incisal-left] to inferior-right [cervical-right] which corresponds to a right oblique orientation after Fox and Frayer (Fox and Frayer 1997). Lightly expressed scratches are also visible on both canines, especially on

their protruding labial surfaces. Both I2s exhibit *in-vivo* enamel chipping on the distal corners and incisal edges (right: 1.74mm x 1.3mm; left: 1.53mm x 1.38mm). Chipping is also present on the disto-buccal corner of RM1 (4.2mm x 2.04mm) and the disto-lingual surface of the LM1 (3.9mm x 1.91mm) (Fig. 6A). Overall, the damaged crown areas are smaller on incisors than on molars, and smaller on the left compared to the right side.

The disto-buccal cervical region of the right M1 and mesio-lingual cervical area of the right M2 exhibit a cylinder-shaped interproximal groove (Fig.7A) indicative of habitual tooth-picking (Margvelashvili et al. 2013). The alveolar crest between the RM1/RM2 is slightly atrophied, having a blunt and flat top. A marked gap exists between the mesial root of RM2 and the surrounding alveolar bone. This is the only radix-alveolar gap in the whole mandible (Fig.7A, B), and indicative of local marginal periodontitis (Margvelashvili et al. 2013).

The cervical circumference of P4s, M1s and M2s is covered with dental calculus (code 2) (Fig. 8A). Calculus deposition on the left side is more expressed than on the right side.

Mandible **D211** is associated with the cranium D2282 (Rightmire et al. 2006) representing a young adult. Overall occlusal tooth wear is moderate (grade 3). Interproximal wear is substantial, as evidenced by flattened mesial and distal surfaces between adjacent teeth. Interproximal wear is most evident between P3/P4, P4/M1 and M1/M2.

D211 exhibits "dental crowding" in the areas of the canines (Fig 3); both Cs are slightly misaligned, with their mesial surfaces turned lingually and distal surfaces rotated labially. In standard (non-crowded) mandibular dentitions, the mesial edge of the canine is in contact with the distal edge of the I2 and the distal edge of the canine is in contact with the mesial edge of the P3. Compared to this standard orientation, the right canine of D211 is rotated by 26°, and the left by 15.5°. Crowding is also present in the area of RM3 (Gabunia et al. 2001); this tooth exhibits marked lingual tipping such that there is occlusal wear on the mesial aspect of its buccal surface. Crowding of the anterior dentition is likely due to mesial relocation of the entire tooth row during eruption of the M3.

The alveolar border of D211 is inferior to the anatomical cervices of the dentition, but root furcations are not visible. In the posterior dentition, the interdental alveolar crests are slightly rounded.

The Cs show light linear enamel hypoplasia (LEH) with the extent of the defect less than 1/3 of the tooth (Fig. 4B). The mesial corner of the LI2 labial surface reveals two right obliquely-oriented parallel scratches. Moderate *in-vivo* enamel chipping is present on the mesial edges of RI1 (0.61mm x 0.44mm) and RI2 (1.09mm x 087mm) (Fig. 6B). The distal corner of LI2 was chipped off *in-vivo* (2.81mm x 1.86mm), and this lesion exhibits smoothened edges. Minimal chipping is also observed on the mid-lingual portion of RM1 (mesial margin of entoconid) (0.83mm x 0.6mm).

Central incisors, LI2, M1s and RM2 reveal remnants of dental calculus around the cervical areas, and mid and incisal portions on approximal surfaces of incisor crowns (Fig. 8B). The alveolar rim around RP3/P4, RP4/M1, RM1 and LP3/P4 exhibits a porous and thickened morphology, which is most expressed in the RP3/P4 area, indicative of moderate periodontitis (Fig. 9).

Mandible **D2600** is associated with the cranium D4500 (Lordkipanidze et al. 2013), and represents a mature adult individual (Fig. 3). Tooth wear is severe (grade 8). The alveolar border is inferior to the anatomical cervices of the dentition. Root furcations are visible on RP3s, RM1 and M2s. The Is, RM1 and M2s are worn down to the cemento-enamel junction (CEJ). There is extensive tertiary dentine formation and pulp chamber exposure on Is, Cs, RM1 and M2s. The alveolar border (interdental crests and alveolar margins) is taphonomically damaged, such that the *in-vivo* condition of the interdental alveolar crests cannot be assessed.

Interproximal wear is substantial, as becomes evident by the flattened mesial and distal surfaces between C/P3, RM1/M2, and M2/M3 and acute lingual tipping of the front dentition. Left I1 and both I2s show superficial, irregular scratches on their inciso-labial surfaces (Fig. 5B) similar scratches are also present on the four incisors and left canine of the associated cranium D4500). Small *in-vivo* enamel chipping can be observed in the disto-lingual area of RC (1.74mm x 0.84mm), and mesio-lingual edge of RP3 (1.77mm x 0.8mm). Chipping is more expressed on the mesial (1.85mm x 1.2mm) and distal (4.35mm x 3.42mm) edges of LP3, and most substantial on the buccal surfaces of the M2s (right: 3.47mm x 0.57mm; left: 4.78mm x 2.65mm). Right M1 exhibits crown-root fracture with pulp chamber exposure in its mesio-lingual area.

All the teeth preserved in the mandible show hypercementosis (Fig. 10) (Margvelashvili et al. 2013). These reactionary structures affect >1/2 of the incisor root surface and 1/3 of the canine root surface. Both roots of premolars are affected to ≥1/3 of the root surface. Molar roots exhibit only minor hypercementosis. Right M1 is hypercementotic on both root tips, while M2s reveal clearly more hypercementosis on the lingual than on the buccal side of the distal root (Fig. 10A), and slight cementum deposition on the mesial root. Hypercementosis on M3s is restricted to the distal roots. Pulp chamber reduction as an effect of dentine deposition is substantial in both radicular and coronal areas (Fig. 10B).

Right M1 and RM2 have developed an interproximal caries. It is located on the occlusal surfaces (~4.6mm in length and ~2.1mm; both teeth combined) and expands in cervical direction on the approximal RM1/RM2 surfaces. The EDJs of both teeth have been compromised, but the carious lesion did not reach the pulp cavities. The depth of the demineralized regions ranges from 0.4 mm (M2) to 0.5 (M1) mm. The caries is moderate to deep, with demineralized dentine and a clearly observable cavity (Fig. 7C, D).

Right I2 and RM1 root apices exhibit radicular lesions, presumably periapical cysts (Margvelashvili et al. 2013). The cysts are spherical and have diameters of ~6.5mm (Fig. 11A,

B). The respective dental root apices are opening into the cyst cavity. Bone remodeling on the internal layers of the cyst is moderate. Both P4s likely were lost *antemortem*, as evidenced by the smooth alveolar bone surface and incomplete bone remodeling of the empty spaces. The alveolar border of the lingual side of RP4 of D2600 is only slightly atrophied (~1.7mm from the alveolar plane) and the buccal cortical wall is completely atrophied down to the level of the roots (~19mm from the alveolar plane). The alveolar socket itself exhibits substantial bone remodeling. The left P4 must have been lost shortly before death as the alveolar socket is only moderately remodeled. The left M1 had been lost *pre* or *postmortem*. The RP4 of the associated cranium (D4500) is overerupted such that its occlusal surface is inferior to the occlusal plane. Overeruption relative to the occlusal plane (as measured on the synchrotron tomography data) is ~2.3mm. Tooth wear on RP4 is less extensive than on its antimere, LP4, because RP4 lacked occlusal contact with its (lost) mandibular antagonist for an extended period of time (the crown of RP4 is 1-1.8mm higher than that of LP4). Estimates for wear-corrected overeruption thus range from 0.50 to 1.3mm. Using modern human population data on overeruption as a reference (Craddock and Youngson 2004; Craddock et al. 2007), this indicates a loss of the mandibular RP4 minimum several months to several years before death (Fig. 11B).

Canines, P3s and M3s exhibit moderately expressed calculus with a patchy and uneven distribution on enamel (Fig. 8C).

The left condyle of the ascending ramus reveals osteoarthritic alterations on the temporomandibular joint (TMJ) surface. The surface is eroded and flattened (Fig. 3), and the condylar head has a reduced overall size (left condyle: ML length: 23.4mm, AP length: 12.7mm, height: 7.8mm; right condyle: ML length: 24.4mm, AP length: 15.4mm, height: 8.0mm). A similar pattern of osteoarthritic alteration is present on the matching surface of the glenoid fossa (Lordkipanidze et al. 2013).

Mandible **D3900** is associated with the edentulous cranium D3444 (Lordkipanidze et al. 2006), representing an elderly individual. The only tooth present *pre mortem* (but lost *postmortem*) was the left canine, as the LC tooth-socket had not undergone noticeable bone remodeling (Lordkipanidze et al. 2005; Lordkipanidze et al. 2006; Margvelashvili et al. 2013). The alveolar process is almost completely atrophied, and the *in-vivo* loss of the alveolar bone reaches a depth of 16mm (Fig. 12). A residual alveolar ridge is only present in the region of the front dentition, where the loss of teeth must have occurred later than in the posterior region (Lordkipanidze et al. 2005; Lordkipanidze et al. 2006) (Fig. 3).

Modern human HG dentognathic pathologies

Both the Australian and Greenlander samples represent nearly all the stages of dental wear (Fig. 2) from none to complete wear-down of the tooth crowns. Both samples also display a wide range of pathologies, from congenital malformations and developmental defects to occlusal

pathologies, infections and *antemortem* tooth loss. Results are summarized in Figures 13, 14 and Table 2.

Australian and Greenlander samples exhibit a similarly wide range of occlusal pathologies. Both samples exhibit distal shift of the permanent lower M1 relative to the upper M1. Enamel hypoplasia is similarly distributed in Australians and Greenlanders. In both populations, enamel chipping is present in individuals with moderate to advanced dental wear. Hypercementosis is weakly expressed in both samples, and only present in individuals with advanced dental wear (grade >4). One of the most consistent dental wear-related changes observed in both populations is pulp chamber reduction**,** starting at dental wear stage 4 (Australians) and 3 (Greenlanders) (Fig. 13A). In Australians, the pulp chamber is gradually reduced until near-closure. In Greenlanders pulp chamber reduction does not reach near-closure. Dental caries was found only in the Australian sample. Cases of caries are equally distributed over the dentition, but restricted to tooth crown surfaces, while roots are not affected. The prevalence of caries is highest in dental wear groups 5 to 6, which is during the period of moderate-to-advanced tooth wear, but before the onset of wear-related *in-vivo* tooth loss. Prevalence is higher on approximal than occlusal surfaces. Pathologic periodontal pockets and local infections are present in both populations, with higher prevalence in Greenlanders than Australians, and appearing at dental wear stages 4 (Australians) and 3 (Greenlanders). Similarly, local infections emerge at dental wear stages 4 (Australians) and 3 (Greenlanders), and their prevalence correlates with dental wear in both populations (Fig. 13B). The prevalence of dental plaque and calculus is higher in Greenlanders than in Australians, with more calculus accumulation per tooth.

Both populations show a complex pattern of periodontal pathologies. Root stripping and furcation involvement was observed from dental wear groups 4 (Australians) and 2 (Greenlanders) onward. The prevalence of these features remains fairly constant over subsequent dental wear stages, but at higher levels in Greenlanders than Australians. Structural changes of the alveolar bone appear at dental wear stage 2 in both groups. It is worth noting here that root exposure also occurs in periodontally healthy populations, and is not necessarily a consequence of periodontal disease, but an effect of continuous eruption (Begg 1954; Kaifu et al. 2003; d'Incau et al. 2012). *Antemortem* tooth loss was observed in both populations, with higher frequencies in Australians than Greenlanders. However, *antemortem* tooth loss starts earlier in Greenlanders (dental wear stage 3) than in Australians (dental wear stage 6) (Fig. 13C). Temporomandibular joint degenerative pathologies are more prevalent in Australians than Greenlanders. This feature appears at advanced dental wear stages (>5).

Comparison of dentognathic pathology patterns in modern human HGs and Dmanisi

Table 2 shows that all the DP features recorded in the comparative HG sample are also found in the Dmanisi hominins. Since the list of features examined here (Table 1) comprises the full range of known modern DPs, this indicates that the diversity of DPs in Dmanisi is similar to that in

modern human HGs. Furthermore, the Dmanisi sample does not exhibit any atypical (non-standard) pathologies that would set apart this fossil sample from the modern human HG samples examined in the study.

Dmanisi clearly differs from the modern HG sample in exhibiting higher levels of enamel hypoplasia, and hypercementosis. At the same time, the Dmanisi sample shares similarities with Australian and Greenlander samples. Infection-related pathologies including carious lesions are present in Dmanisi and the Australian sample, but absent in the Greenlander sample. On the other hand, occlusal pathologies (rotation and tilt) are found in Dmanisi and Greenlanders, but not in Australians. The prevalence of enamel chipping is highest in the Greenlander sample, moderate in Dmanisi, and lowest in the Australian sample.

Figure 13 shows correlations between dental wear and DPs in the modern human HG and Dmanisi samples. In all three groups, pulp chamber reduction, local infections, *antemortem* tooth loss and subsequent alveolar bone remodeling show significant correlation with tooth wear. The slopes of group-specific regression lines were compared with resampling statistics (see Methods). Figs. 13 D-F shows marked differences between population samples, indicating population-specific rates of incidence (number of new DP cases per dental wear stage) of these pathologies. Rates in the Dmanisi sample tend to be high, but within the variation of modern human rates.

Figure 14 presents the results of a resampling test that addresses the question: What is the probability to see a "Dmanisi-like" DP pattern in a modern HG population sample? Resampling was used to draw specimen triplets (two early-stage dental wear specimens, matching D2735 and D211, and one late-stage specimen, matching D2600) from the modern HG samples. To compare DP patterns among specimens triplets, we calculated the distance metric *D* measuring dissimilarity across 11 DP features (see Methods). *D*-distributions were evaluated for the comparison of Dmanisi with Australian and Greenlander triplets, as well as for comparisons within the modern HG samples. Among-Australian *D*-values are comparatively small, among Greenlander values are moderate, and Dmanisi-to-modern values are large. The DP pattern of the Dmanisi triplet is at the upper end of variation, or outside the range of variation, of the DP pattern seen in corresponding modern HG triplets. These results indicate that the prevalence of DP features in Dmanisi tends to be higher than in modern HG samples with similar dental wear stages.

Together, these observations permit a formal test of the hypotheses proposed earlier:

H01 accepted: The diversity of dentognathic pathologies is largely similar in modern HGs and Dmanisi.

H02 falsified: Our data show that there are clear population-specific differences in the prevalence of dentognathic pathologies. The Dmanisi sample shows the highest prevalence relative to stage of dental wear.

H03 partially accepted/falsified: Dentognathic pathologies tend to accumulate at statistically similar rates in Dmanisi compared to modern HG samples. However, the Dmanisi sample differs from the modern HG samples in several respects: at young age (as represented by the adolescent individual D2735) tooth wear is already relatively advanced, and pathologies are comparatively prevalent. When dental wear is used as a population-specific proxy of individual age (see discussion below), our data indicate that, in all three populations, dentognathic pathologies tend to accumulate with increasing age, but probably at population-specific yearly rates.

DISCUSSION

Falsification of H02, and partial falsification of H03 indicates that various factors contribute to population-specific differences in the formation of dentognathic pathologies. These will be considered in greater detail in the following section.

While our data indicate that tooth wear is an important cause of DP, it has to be kept in mind that it is one of many factors (Fig. 1). Similar results were found in Late Pleistocene humans, where all pathologies but caries correlate with dental wear (Lacy 2014a). The development of DP must be seen as a multifactorial process, where the effects of exogenous and endogenous factors such as environment, technocultural behavior, genes and development accumulate over an individual's lifetime. Moreover, each specific pathology can trigger the formation of various other pathologies, such that they are linked to each other via a network of cause and effect.

The Dmanisi and modern HG samples display largely similar patterns and incidence of dentognathic pathologies, but marked differences in prevalence (Fig 13, 14, Table 2). We thus surmise that the basic functional/biomechanical morphology of the dentognathic system, and its "disease trajectory" (incidence) is similar in all species of the genus *Homo*, but that there are species-specific differences in "disease load" (prevalence). Our study is in accordance with the recent results of Lacy (2014a) indicating that late Pleistocene hunter-gatherers suffered from similar oral diseases as modern humans, but with population-specific prevalence. As shown earlier (Margvelashvili et al., 2013), dental wear-induced compensatory mechanisms such as continuous dental eruption, mesial drift and lingual tipping are also similar among the groups studied here, indicating common response mechanisms to dentognathic aging and pathology in *Homo*.

Differences between the populations studied here indicate differences in the factors influencing the rate of formation of pathologies. The absence of dental caries in Greenlanders is probably related to two factors: a diet with a low proportion of carbohydrates and a high rate of dental wear resulting in the elimination of cariogenic regions. Significant accumulation of dental

calculus in Greenlanders is likely related to their highly specialized dietary habits. Plaque formation is typically counteracted by hard food particles, which are abundant in plant-rich diets. The low proportion of fruit and vegetables but high proportion of meat in the Greenlander diet might have prevented the natural cleansing activity of hard food on the crown circumference (non-occlusal surfaces) of the teeth, resulting in a high rate of calculus accumulation. On the other hand, the comparatively broad spectrum of pathologies observed in Dmanisi and in the Australians indicates more generalized dietary habits.

The subadult and young adult Dmanisi specimens D2735 and D211 exhibit a high prevalence of pathologies (Fig. 3) compared to HG individuals exhibiting similar dental wear stages (at which HG individuals are fully adult). The high prevalence of enamel hypoplasias indicates stress during infancy and early childhood, probably related to food shortages and infectious illnesses. The high prevalence of periodontal pathologies in Dmanisi already during subadult and early adult life is indicative of early onset of pathogenic processes, suggesting a generally higher levels of stress on the dentognathic system than in modern HG populations. The high prevalence of pathologies in the D2600 mandible compared to modern HGs at the same dental wear stage is also indicative of an "early-onset" pattern of DP in Dmanisi. Furthermore, complete tooth loss in the D3900 mandible is most likely a consequence of multiple DP.

In the following sections we interpret each dentognathic pathologic feature in the light of what is currently known about underlying cause-effect networks.

Congenital diseases

Developmental defects, agenesis

The processes leading to enamel hypoplasia (EH) are only partially understood (Goodman 1989 ; Hannibal and Guatelli-Steinberg 2005; Brook 2009; Michael et al. 2010). Enamel hypoplasia likely reflects episodes of nutritional deficiency and/or infectious disease, and is thus considered a developmental stress marker (Guatelli-Steinberg 2001). The occurrence of LEH strongly depends on the general health status of an individual (Ogden et al. 2007). As an effect, no two individuals exposed to similar environmental/nutritional stress will show the same pattern of enamel hypoplasia expression (El-Najjar et al. 1978).

Australian and Greenlander samples exhibit similar incidence of enamel hypoplasia, indicating higher levels of stress during the time of dental crown formation. The hypoplastic defects are most frequently located in the cervical thirds of the tooth crowns, and the canines are the most affected teeth in Australians, whereas in Greenlanders mostly postcanine teeth are affected in their cervical thirds. The stress thus seems to have occurred at individual ages between 3.0 and 4.5 years in Australians, and between 2.5 and 5.0 in Greenlanders (age estimation after Rose et al. 1985), which largely corresponds to the weaning time of HG populations (Konner 2005; Marlowe 2005). Enamel hypoplasia defects in D211 are mild, but severe in D2735. In the latter

specimen, EHs co-occur on homologous (corresponding L/R) teeth, and are concentrated on the cervical areas of tooth crowns, indicating systemic stress events during late stages of crown formation (Jalevik and Noren 2000). Recalling that crown formation of Is, Cs, and M1s starts in utero, and of Ps and M2s starts in early infancy, the cervical EH pattern indicates recurrent stress events. Applying Neanderthal and modern human tooth formation standards (Guatelli-Steinberg et al. 2014), recurrent stress events occurred during late infancy or early childhood (Neanderthals: ~3.1, modern humans: ~3.4-4.55 years) and might thus be related to weaning. Applying presumed *H. erectus* standards (Dean et al. 2001), weaning-related stress in D2735 might have occurred even earlier.

The paleoclimate at Dmanisi was temperate with harsh winters (Messager et al. 2010; Blain et al. 2014), implying possible seasonal food shortage and nutritional stress affecting the amelogenesis in the infants' developing dentition. Differences between D2735 and D211 in the severity of hypoplastic defects may indicate individual differences in susceptibility of amelogenesis to nutritional stress, or a harsher winter during the infancy of D2735 compared to D211.

Bilateral and unilateral agenesis of M3s is a relatively prevalent condition (2-41%; (Alam et al. 2014) in modern human populations and considered to represent a developmental variant rather than a pathology (Vastardis 2000; Arte and Pirinen 2004; Tunc et al. 2011; Alam et al. 2014). Because M3 formation is initiated late during ontogeny, it is potentially under stronger influence of jaw expansion than the formation of M1 and M2 (Scheuer and Black 2000; Zoetis et al. 2003). Reduced posterior expansion – be it as an effect of evolutionary adaptation or *in-vivo* modification – thus may result in spatial constraints that compromise M3 formation.

D2735 exhibits agenesis of LM3, while D211 exhibits bilateral M3 reduction, both in terms of tooth size and available alveolar space. M3 agenesis has also been observed in the Omo 75-14 a+b specimen (right M3), and on both sides of the KNM-WT15000 mandible (Wallace 1977; Dean and Smith 2009). We suppose that, in evolutionary terms, the joint effect of facial size reduction and space limitation in the retromolar area resulted in M3 size reduction and/or agenesis already in early *Homo*.

Tooth wear and dentognathic pathologies

Patterns and rate of dental wear depend on the type of food, food preparation techniques, and the involvement of the dentognathic system in non-masticatory activities. Food-related behaviors tend to be socially transmitted within populations (Molnar 1971; Smith 1972). Differences in dental wear patterns between populations are thus not only diet-related but indicate technocultural differences. Furthermore, environmental factors such as dust and grit particles contaminating the food influence wear rates substantially (Jokstad et al. 2005; Lucas et al. 2013). Massive dental wear can result from a combination of high masticatory loads, abrasive food, the presence of grit, and/or a long duration of exposure to these factors (i.e., old age).

We suppose that the wear pattern of the Dmanisi mandibles reflects a common baseline (diet and paramasticatory activities), as well as individual (genetic and behavioral) factors. The mildly worn posterior dentition of the young adult D211 mandible contrasts with the moderately worn M1s of the adolescent D2735 mandible, indicating individual differences in dental eruption timing, mastication and/or paramasticatory activities, as they are also found in modern populations. The severe tooth wear and tooth loss observed in the adult D2600 mandible, and complete tooth loss in the edentulous mandible D3900 may also have a multifactorial origin. Likely factors are an abrasive diet, extensive paramasticatory activities, as well as a long duration of wear, suggesting an advanced age.

In the D2735 mandible, tooth wear on the right side is more pronounced than on the left side, while the accumulation of dental calculus is more pronounced on the left than on the right side. This asymmetric pattern suggests a right-sided mastication preference: the preferred side is more exposed to dental wear, and hard food items and saliva tend to act as a natural cleansing system preventing the deposition of calculus. Conversely, the opposite side is less exposed to dental wear and hard food items, resulting in more calculus deposition. Further evidence for right-side masticatory preference comes from the observation that enamel chipping defects on that side are larger than on the left side. The preference for right-side mastication is possibly related to an occlusal anomaly on the left side resulting from maxillary LP4 malformation/malorientation. This condition might have resulted in discomfort and/or low efficiency when masticating on the left side, such that the right side was preferred by this individual.

Stripped anatomical cervices and root furcation visibility are typically interpreted as indicators of generalized periodontal disease (Hillson 2002; Vodanović et al. 2012). However, anatomical cervices can become visible even in periodontally healthy individuals, mostly as an effect of continuous dental eruption compensating for advanced tooth wear (Levers and Darling 1983; Clarke and Hirsch 1991; Danenberg et al. 1991; Glass 1991; Kaifu et al. 2003; Hillson 2008; Margvelashvili et al. 2013).

Striations of the labial surface of the front teeth are related to paramasticatory activities and handedness of the individual (Fox and Frayer 1997). For example, striations in right oblique direction are considered to be an effect of “stuff-and-cut” techniques (fixing an item between the front teeth, pulling it with the left hand, and cutting with a sharp tool held by the right hand) (Brace 1975; Bax and Ungar 1999; Molnar 2008). All of the Dmanisi mandibles show striations on the labial surfaces of the incisors, and to a lesser extent on the canines. In D2735 and D211, striations mostly have right oblique orientation, likely suggesting right-handed stuff-and-cut actions. The front dentition of D2600, which is worn down to labially rounded root stumps and exhibits pronounced irregular striations, reflects continuous and intense para-masticatory activities. Taphonomic evidence indicates that tool-mediated food preparation and meat consumption belonged to the behavioral repertoire and dietary niche of the Dmanisi hominins (Lordkipanidze et al. 2007; Lordkipanidze et al. 2013; Margvelashvili et al. 2013).

Enamel chipping occurs when the bite force pressure surpasses the strength of the enamel. Chipping is independent of the size of the foreign object (grain of sand, bone, etc) (Scott and Winn 2011), and tends to occur when an object exerts high local forces near the occlusal edge of a tooth (Constantino et al. 2010).

The high prevalence of enamel chipping in the Greenlander sample is in concordance with previous studies documenting high frequencies in arctic populations and suggesting a causal link with hard-object mastication and paramasticatory activities (Pedersen 1947; Turner and Cadien 1969; Scott and Winn 2011). Like in the modern comparative sample, the frequency of enamel chipping in Dmanisi increases with tooth wear. This pattern is indicative of hard-object consumption and/or paramasticatory activities, resulting in accumulation of chips over an individual's lifetime. It is worth noting that dental microwear data of D211 and D2735 (Pontzer et al. 2011) and additional data of D2600 (personal observation) indicate that the *antemortem* diet of these individuals was not exceptionally hard or tough (Pontzer et al. 2011). As exemplified by the following considerations, enamel chipping seems to have been a rare traumatic event: Modern human masticatory activity comprises about 1’000’000 chewing cycles per year (Verplancke et al. 2011). Assuming that the “lifespan of masticatory activity” of Dmanisi D2735, D211, and D2600 was 10, 20 and 30 years, respectively, one chipping event happened in 2.5, 5, and 4 years. Accordingly, the average incidence in Dmanisi was 1 chip per 3’800’000 chewing cycles. The actual incidence in D2600 might have been higher, given the fact that early chips have been effaced as an effect of dental wear.

Hypercementosis results from excessive continuous cementum deposition during an individual's lifetime. It is one of the main mechanisms of continuous dental eruption and is thought to be triggered by heavy occlusal loading (Pinheiro et al. 2008). Although both populations show evidence of hypercementosis, it is at a low level of expression. Greenlanders are known to bear heavy occlusal loads during paramasticatory activities, especially on the anterior teeth (Pedersen 1947; Spencer and Demes 1993; Clement et al. 2012), such that one would expect substantial hypercementosis. In fact, an earlier study reported “pronounced absorption of the apical part of the roots with hyperostosis of the remaining part” in Greenlanders (Pedersen 1947) p.24, which was later formulated as “considerable resorption with compensatory hypercementosis” (Pedersen 1949) (p.175, plate 27, Fig. 150b). However, hypercementosis is an age-related response to heavy masticatory load (Pinheiro et al. 2008) whereas root resorption is an effect of local occlusal overload (Schätzle et al. 2005). Hypercementosis is thus more likely to be the cause than the effect of root resorption (Schätzle et al. 2005). These observations indicate that caution is warranted when interpreting population-specific differences in hypercementosis in terms of different masticatory regimes.

Given the advanced degree of tooth wear in the D2600 mandible (Margvelashvili et al. 2013), hypercementosis in this individual appears as a compensatory mechanism to anchor the lingually tipped front dentition (note more intense cementum deposition on the lingual side; Fig. 10A) and

to provide support for the overerupted dentition. The less worn teeth of the D211 and D2735 mandibles are free from such modifications, thus supporting the notion that hypercementosis in the Dmanisi sample is related to advanced dental wear.

Pulp chamber reduction results from continuous dentine deposition on the surface of the pulp cavity. This age-related pattern is well-documented in modern human populations (Prapanpoch et al. 1992; Kvaal et al. 1995; Murray et al. 2002). Rates of dentine deposition depend on the tooth type and the location within the tooth (crown/root) (Murray et al. 2002), continuous deposition ultimately leads to near-closure of the pulp chamber. Rates of wear-related pulp chamber reduction are largely similar in the HG populations studied here (Fig. 13A), but reduction commences at earlier dental wear stages in Greenlanders. This might reflect a population-specific response to heavy masticatory loads [mechanical stress is known to induce odontoblast differentiation, thus reducing the pulp chamber size (Lee et al. 2010)]. Pulp chamber reduction in the D2600 teeth is substantial, but absent in D2735 and D211. Using the modern human pattern, where pulp chamber reduction is tightly correlated with dental wear as a yardstick, D2600 thus appears as an individual of advanced age compared to D211.

Caries is one of the most widespread chronic diseases among modern human populations and is attributed to frequent consumption of food rich in fermentable carbohydrates (Selwitz et al. 2007). Caries has been documented in many Late Pleistocene Neanderthal and human populations, indicating that the prevalence of this condition was more significant than previously assumed (Lacy 2014). The highest prevalence (51%) of caries was recorded in the population of Taforalt, Morocco (~14000 BP), and was interpreted as reflecting a high percentage of starchy plant foods in their diet (Humphrey et al. 2014).

In Plio-Pleistocene hominins, however, caries has been documented so far in only a few cases (Clement 1956; Puech et al. 1980; Grine et al. 1990; Bartsiokas and Day 1993). The scarcity of cases possibly reflects a low actual incidence of caries, be it as an effect of non-cariogenic diet and/or food preparation, or as an effect of heavy dental wear, which tends to eliminate early cariogenic areas on occlusal surfaces (Maat and Van der Velde 1987; Meiklejohn et al. 1992). Alternatively, the actual incidence of caries might be higher than currently estimated, because small to medium-sized carious defects tend to be overlooked in fossil specimens, especially if they are stained as an effect of the fossilization processes. A revision of the Plio-Pleistocene fossil hominin record is required to clarify this issue.

The interproximal carious lesion in the D2600 mandible [which was overlooked in an earlier DP analysis of this specimen (Martín-Francés et al. 2013)] is at a typical location in the sense that the interproximal space is most prone to the formation of bacterial plaque-mediated caries (Selwitz et al. 2007), and least accessible to wear-related elimination of the cariogenic region.

In the Australian sample studied here the prevalence of carious lesions increases toward DW stages 5 and 6, then decreases with advanced DW. The "bell-shaped" curve of prevalence is most probably due to the combined effects of caries accumulation and dental wear over time. Increased wear may result in effective elimination of carious regions, while carious teeth may be lost as an effect of caries-induced pathologies. A similar “bell-shaped” caries distribution was observed by Maat and Van der Velde (1987), where the highest prevalence of caries was recorded in individuals with light-to-moderate dental wear. The diet of Australian aboriginal populations has been reported to be low in cariogenic substances (Davies et al. 1997), but caries prevalence is comparatively high (from 15 to 48%) (Barrett and Williamson 1972; Schamschula et al. 1980; Pascoe and Kim Seow 1994). The comparatively low prevalence of caries in Ngaraangbal tribes was related to a high proportion of marine foods (Elvery et al. 1998). Marine diets are thought to be cariostatic due to increased fluoride levels, and due to the antibacterial effects of increased alkalinity in the oral cavity (Sealy et al. 1992; Oxenham and Matsumura 2008).

Contrasting with Australians, the Greenlander sample studied here is free of caries. The lack of caries in Arctic populations has been reported previously; no caries was found in Aleuts, and a prevalence of less than 1-5% in Greenlanders subsisting on a traditional diet (Pedersen 1947; Keenleyside 1998). The absence or low prevalence of caries in these populations had been attributed to attrition-related elimination of the caries-prone areas, as well as to the specific diet, which lacks refined sugars and starches (Keenleyside 1998). On the other hand, the high prevalence of caries (14.4%) in the Ipiutak sample from Point Hope has been related to dietary specialization; this population mostly subsisted on caribou, and to a lesser degree on marine mammals (Costa 1980b; Oxenham and Matsumura 2008).

The pathologic pocket in the D2735 mandible is likely a result of repeated toothpicking between RM1/RM2, which compromised the mucosal surface of the gingival sulcus. This lesion opened a path for microorganisms, provoking the detachment of the periodontal ligament, a spread of the infection, and the resorption of the interdental alveolar crest (Fig. 7A, B). This process eventually resulted in local marginal periodontitis (Margvelashvili et al. 2013).

Radicular lesions in D2600 resulted in periapical cyst formation (Rosenberg et al. 2010), resorption of the affected periapical bone around RI2 and RM1, and destruction of the adjacent external cortical plate of the mandibular corpus (Margvelashvili et al. 2013). As stated earlier (Margvelashvili et al. 2013) this condition is a consequence of excessive tooth wear. D2600 completely or nearly completely wore off the crowns of all four incisors, Cs, RM1 and M2s, which led to massive dentine and even pulp chamber exposure. This condition entailed chronic root canal infection and apical periodontitis, which ultimately developed into cystic apical periodontitis, a process that is clinically well-documented in modern humans (Nair 2004; Dag Orstavik and Ford 2007). Similar patterns have been found in HG populations from Sweden and

Siberia (Lieverse et al. 2007; Molnar 2008; Molnar 2011), as well as in Natufian HG populations (Eshed et al. 2006).

The rate of calculus accumulation depends on the alkalinity of the oral environment, which increases the absorption of minerals from the oral fluids (saliva and gingival cervic fluid) (Arensburg 1996; Lieverse 1999; Jin and Yip 2002). Calculus formation is positively affected by protein-rich diets increasing the oral alkalinity (Hillson 1979). Plaque and calculus formation also depends on the health status of the gingiva, which, in turn, is influenced by the general health status of the individual (Garcia et al. 2001). Dental plaque tends to accumulate on teeth surfaces adjacent to areas affected by gingivitis, resulting in a higher incidence of calculus than in healthy regions (Ramberg et al. 1995; Garcia et al. 2001). The location of dental calculus in the Dmanisi mandibles suggests that it was of supragingival origin. This type of calculus occurs both in healthy and periodontally diseased individuals (Lieverse 1999). Calculus accumulation is most intense in D2735, possibly indicating a low flow of saliva (Humphrey and Williamson 2001). Given the young age of D2735, and its extended list of DP the general state of health of this individual was probably more compromised than that of the adult individuals.

Periodontitis is one of the most prevalent pathological conditions of modern human populations (Pihlstrom et al. 2005), and has been related to plaque accumulation, virulence of the plaque biofilm, microbiota, immunity factors, individual susceptibility and even gender (Albandar and Rams 2002; Nishihara and Koseki 2004; Merchant et al. 2005). Generalized periodontitis causes alveolar crest destruction, which leads to deep furcation involvement, reduction of dento-alveolar contact and the creation of rounded, porous margins of the alveolar bone. Eventually, a gap forms between the tooth and the alveolar crest, thus loosening dental fixation (Ogden 2008). Periodontitis is also well documented in the fossil hominin record (Grine et al. 1990; Gracia-Téllez et al. 2013; Lozano et al. 2013).

Antemortem tooth loss is a consequence of the eventual breakdown of the dentognathic structures as a long-term consequence of dental wear, infections and/or trauma. The sequence of tooth loss in modern human populations follows a specific pattern. Tooth loss typically starts in the posterior dentition, while the mandibular anterior teeth are the last to be lost (Marcus et al. 1996). Wear-related tooth loss in the Australian sample follows this sequence. However, a substantial proportion (40%) of the Greenlander sample exhibits primarily anterior tooth loss, which corresponds to a pattern that has been related to the excessive paramasticatory anterior tooth use (Merbs 1968).

The posterior-to-anterior pattern is likely related to the higher incidence of DP in the posterior relative to the anterior dentition; compared to incisors and canines, molars and premolars are more susceptible to dental infections such as caries because their cusp and fissure topography is more complex (Selwitz et al. 2007). Also, masticatory loads are higher in the posterior section of the mandible, resulting in higher degrees of wear, and earlier exposure of the pulp chamber.

Once a tooth has been lost, resorption of the respective alveolar bone is extensive during the first 6 months, then slows down and lasts at least 2 years until complete resorption of the residual alveolar ridge (Zmyslowska et al. 2007; Van der Weijden et al. 2009). Clinical studies show that during the first 6-7 months the alveolar ridge is reduced in height by 3.0-3.3mm, and in width by 2.94-4.5mm (Lekovic et al. 1998; Barone et al. 2008). Complete residual ridge resorption results in a height loss of 15mm in the anterior and posterior regions of the mandible (Cawood and Howell 1988; Floyd et al. 1999).

Both P4s of D2600 were lost *antemortem*. The right P4 must have been lost relatively long before the individual's death, as the alveolar socket is remodeled and the maxillary antagonist P4 is overerupted ((Lordkipanidze et al. 2013) see Fig 1G) and bears less occlusal wear than its antimere. In modern human populations the mean dental overeruption due to missing antagonists was reported to be ~0.8mm (SD 0.65mm) within an interval of >10 years (Christou and Kiliaridis 2007). Other studies suggest that overeruption may reach 2 to 5.4mm (Kiliaridis et al. 2000; Craddock and Youngson 2004). Based on these comparative data, the loss of RP4 in D2600 may have happened around 5 to 10 years prior to death. Since the D2600 individual had an open bite (Lordkipanidze et al. 2013), the P3/P4s were the first teeth to be in occlusal contact during mastication. High masticatory and paramasticatory forces may have overloaded the periodontal structures, leading to eventual tooth loss.

In D2600 the alveolar border of the lingual side of RP4 is only slightly resorbed but the buccal cortical wall is completely resorbed down to the level of the root apex. This pattern of asymmetry indicates that tooth loss was due to several causes: 1) trauma due to open-bite masticatory overload on the thin buccal cortical wall around the Ps (Katranji et al. 2007; Tal et al. 2012), 2) infection-related tooth loss and 3) wear-related lingual dislocation of Ps with roots tilted buccally (Fig. 11B), followed by resorption of the buccal cortical wall (Clarke and Hirsch 1991).

The D2600 mandible displays a relatively early stage of alveolar bone resorption, while the D3900 mandible represents the final stage. D3900 lost at least 16mm in height of the alveolar bone (Fig. 3 and 12), while D2600 lost around 19mm of the buccal alveolar bone (Fig. 11B). Comparative data from modern human populations (Lekovic et al. 1998; Kiliaridis et al. 2000; Christou and Kiliaridis 2007; Barone et al. 2008) permit to provide minimum estimates of the "edentulous life span" of the D3900 individual. Assuming ~0.25-0.65mm of alveolar bone resorption per month of edentulous life [calculated from external and internal vertical wall loss over a period of 6-7 months (Lekovic et al. 1998; Barone et al. 2008)] results in a minimum of ~26-64 months of edentulous life.

Evidence that the D3900 individual survived without teeth for at least 2.6 years supports the hypothesis that even the complete breakdown of the dentognathic system was unlikely to limit

the lifespan of early *Homo* at Dmanisi, since the impaired dentognathic masticatory function was compensated with tool-mediated soft food preparation (Lordkipanidze et al. 2013).

Temporomandibular joint disorders are among the most prevalent age-related pathologies of the dentognathic system (Zhao et al. 2011), and are typically caused by mechanical overload of the joints (Machon et al. 2011). However, a single traumatic incident can also provoke the pathology (Zhao et al. 2011). Temporomandibular joint disorders in the HG populations studied here are clearly related to dental wear (and thus age). The development of TMJ osteoarthritis in D2600 is likely caused by masticatory overload. The right side of the associated cranium D4500 reveals a healed fracture of the zygomatic arch (Lordkipanidze et al. 2013). The fracture probably led to impaired masticatory function on the right side, and consequently, to masticatory overload on the non-affected left side during the healing process, and later on during the individual's life.

Implications for the paleobiology of the Dmanisi deme

The major contrast between the Dmanisi paleodeme and the modern HG populations studied here is that Dmanisi exhibits a higher prevalence and earlier onset of dentognathic pathologies. This pattern is not a consequence of higher tooth wear rates in Dmanisi, as evidenced by the fact that wear-related incidence of DPs in Dmanisi is statistically similar to the incidence in the HG populations. In other words, although tooth wear in Dmanisi is already extensive at early ages, the prevalence of DP is higher than expected for that degree of wear in the HG populations. This pattern permits several inferences on the paleobiology of the Dmanisi deme. Since such inferences largely depend on our current empirical knowledge about cause-effect relationships between various factors and DPs, and on our current knowledge about DP patterns in other Plio-Pleistocene hominins, these aspects will be discussed beforehand.

As indicated in Figure 1, differences in DP patterns can be related to three key factors influencing the (paleo-)biology of a population: diet, culture, and environment. The influence of each of these factors on oral health has been assessed in various comparative studies, both on fossil hominins and extant humans. Dietary cause-effect relationships are best documented for caries, the prevalence of which is clearly related to consumption of starch-rich foods, both in Late Pleistocene and modern (archeological) human populations (Fujita 2012; Da-Gloria and Larsen 2014; Humphrey et al. 2014; Lacy 2014a). As indicated by Costa et al. (1980b), the prevalence of caries may also be related to other dietary preferences not related to starchy diets. Causal relationships between diet and DP patterns other than caries are less clear, although differences and similarities in the prevalence of DPs are often interpreted in terms of dietary differences and similarities, respectively (Lillie 1996; Lacy 2014a; Miller et al. 2014; Flensborg 2015). The dietary shift characterizing the neolithic transition is typically correlated with shifts in DP patterns (Larsen 1995; Lukacs 2008). However, in the transitional Natufian populations, no evidence was found for a shift in oral health, probably indicating that diets consumed by the Natufian HGs and Neolithic agriculturalists did not differ in starch contents (Eshed et al. 2006).

Various studies have established links between population-specific differences in DP patterns and technocultural differences. The neolithic transition best documents culture-mediated shifts in DP patterns (Larsen 2002; Steckel et al. 2002; Roberts and Manchester 2007; Lukacs 2008). Food preparation techniques such as the use of tools, cooking, paramasticatory activities, etc., have been shown to influence the mechanical properties of the food items and their interaction with the dentognathic system, and thus on the patterning of DPs (Barker 1975; Costa 1980a; Larsen 1995; Wrangham et al. 1999; Pereira et al. 2006). At the same time, cultural changes entail changes in the dietary spectrum, such as e.g. the inclusion of meat in the diet of early *Homo* (Lordkipanidze et al. 2005; Lordkipanidze et al. 2007; Braun et al. 2010; Ungar 2012). Furthermore, various studies have established links between antemortem tooth loss (AMTL) and cultural behaviors (Costa 1980a; Lukacs 2007). The prevalence of AMTL increases from early to late Upper Paleolithic populations of *Homo sapiens*, a pattern that is typically interpreted as an effect of culturally mediated increase in lifespan (Oztunc et al. 2006; Lacy 2014a), and also discussed in the context of "biology of care" (Lordkipanidze et al. 2005; Tilley 2012). While these studies indicate positive effects of culture ("cultural buffering") on oral health, it has to be kept in mind that culturally-mediated behaviors can also result in its deterioration. For example, dental ablation/avulsion (Durband et al. 2014), paramasticatory activities, and dietary shifts related to agriculture clearly lead to impairment of the dentognathic system (Molnar 1972; Barker 1975; Larsen 1995; Molnar 2011).

The effects of environmental factors on oral health have been documented in several studies (Sealy et al. 1992; Guatelli-Steinberg 2001; Petersen et al. 2005; Lucas et al. 2014). Lucas et al. (2013) proposed that the presence of quartz dust in the environment (and thus in the diet) substantially affects rates of tooth wear. On a larger scale, climatic conditions (latitude) and climatic fluctuations have been shown to influence the prevalence of dentognathic pathologies, both among modern humans and Neanderthals (Skinner 1996; Larsen 1997; Guatelli-Steinberg 2001; Lacy 2014a).

Comprehensive studies that cover a wide spectrum of DPs in early hominins are currently unavailable, such that the evidence provided in this study can only partially be compared in a broader context. Based on the existing information from the fossil hominin case studies mentioned earlier [see introduction, discussion, and (Ward et al. 1982; Ripamonti 1988; Grine et al. 1990; Gibson and Calcagno 1993)] it is not possible to infer the prevalence of specific DPs. Nevertheless, it appears that the range of DPs in Pliocene to early Pleistocene hominins was similarly wide as in Dmanisi. Furthermore, the available evidence from Plio-Pleistocene homininis suggests that periodontal disease was more prevalent in *Australopithecus* and *Paranthropus* than in early *Homo*, but that caries was more prevalent in the latter taxon. Also, it appears that these differences are related to differences in diet and technoculture, but not to environmental differences. While these hypotheses remain to be tested by detailed comparisons

of fossil specimens, they serve as a guide to draw inferences from DP patterns on the paleobiology of Dmanisi.

The high prevalence of linear enamel hypoplasia in the Dmanisi paleodeme indicates strong environmental effects, probably related to substantial seasonal fluctuations compared to the environment of African early *Homo*. However, the prevalence of DPs in Dmanisi does not seem to exceed the (inferred) prevalence in African early *Homo*, nor the prevalence documented for several DPs in the Neanderthals (Skinner 1996; Guatelli-Steinberg 2004; Guatelli-Steinberg et al. 2004; Lacy 2014a). This indicates that the Dmanisi people were well able to cope with their harsh environment, most probably through technocultural adaptation. The broad range of DPs in Dmanisi likely reflects a broad dietary range, including starch-rich plant foods, and a substantial proportion of meat. The survival of a toothless individual indicates that cultural buffering played an important role. On the other hand, evidence for extensive deterioration of the dentition through paramasticatory use indicates the adverse effects of technoculture on oral health.

CONCLUSION

The quantitative comparative analysis of dentognathic pathologies in the Dmanisi hominins and in samples representing hunter-gatherer populations from Australia and Greenland revealed commonalities and differences between populations: 1) The range of dentognathic pathologies seen in the Dmanisi hominins is similar to that seen in the modern HG samples; these pathologies comprise the full range dentognathic pathologies reported in clinical dentistry. 2) *In-vivo* pathologies are mainly related to heavy masticatory loading, severe tooth wear, and bacterial infection. 3) Within each HG population sample there is considerable inter-individual variation in type and degree of pathologies. Variation tends to be higher in the Greenlander than in the Australian sample. 4) Spatial and inferred temporal patterns of dentognathic pathologies of the Dmanisi hominins differ from those of Australians and Greenlanders in several respects. The comparatively high prevalence of pathologies in the subadult and young adult Dmanisi specimens (D2735, D211) indicates that their dentognathic system was exposed to higher stresses, and at earlier ages, compared to modern HG populations.

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Figures and Tables.

Fig. 1 Network of dentognathic pathologies and etiological factors.

Fig. 2 Frequency distribution of dental wear classes in Australian, Greenlander and Dmanisi populations.

Fig. 3 Dmanisi mandibles. Occlusal views, and graphical representation of dentognathic pathologies. Grayscale coding: unworn (white) to heavily worn (black) teeth.

Fig. 4 Linear enamel hypoplasia in Dmanisi mandibular teeth. A: D2735 LP3; B: D211 LC. Arrows indicate hypoplastic bands.

Fig. 5 Labial striations on Dmanisi mandibular teeth. A: Isolated RI2 (D2854) belonging to mandible D2735; B: LI2 of mandible D2600.

Fig. 6 Enamel chipping in Dmanisi mandibles. A: D2735 RM1 lingual view; B: D211 LI2 labial view; C: D2600 LP3 oblique occlusal view. Arrows indicate chipped enamel.

Fig. 7 Infectious and mechanical dentognathic pathologies in the Dmanisi mandibles. A: D2735 RM1/RM2 cylinder-shaped gutter from toothpick utilization; arrow indicates alveolar crest resorption and cylinder-shaped gutter; white background used for outlining the interdental spaces; B: D2735 RM2 coronal cross section; arrow indicates marginal periodontitis on buccal side (mesiodistal view; synchrotron tomography data, isotropic voxel size 45.7^3 mm^3); C: D2600 RM1/RM2 dental caries; D: D2600 RM2 coronal cross-section of carious lesion (distomesial view; synchrotron tomography dara, isotropic voxel size 45.7^3 mm^3); arrow indicates demineralized carious region on RM2.

Fig. 8 Dental calculus in Dmanisi mandibles. A: D2735 LM2 lingual view; B: D211 RM1 buccal view; C: D2600 RM3 buccal view. Arrows indicate the presence of calculus.

Fig. 9 Mandible D211. Right lateral view. Arrow indicates moderate periodontitis.

Fig. 10 D2600 reactionary structures. Inset figures indicate the location of cross-section (synchrotron data, voxel size $45.7^3 mm^3$). A. RM2 region, coronal section, mesiodistal view; B. Anterior region, labiolingual view. Arrows indicate hypercementosis and pulp chamber reduction.

Fig. 11 Mandible D2600, radicular lesions and *antemortem* tooth loss. A: Anterior view, RI2 radicular lesions (periapical cyst); B: Right lateral view, RM2 radicular lesion (periapical cyst) and RP4 *antemortem* tooth loss. Arrows indicate radicular lesions and *antemortem* tooth loss.

Fig. 12 D3900 mandible. Alveolar bone resorption measure.

Fig. 13 Dentognathic pathology as a function of dental wear in Australian and Greenlander HG and Dmanisi samples. A: Pulp chamber reduction; B: Local infections; C: Number of teeth lost *antemortem*. Red symbols and regression lines: Dmanisi hominins [squares, from left to right: D2735 (DW2), D211 (DW3), D2600 (DW8), D3900 (DW9); Black symbols: *H. sapiens* Australia [normal triangles and solid regression line] and Greenland [inverted triangles and dashed regression line]. Group-specific distribution of regression slopes evaluated by resampling: D: Pulp chamber reduction; E: Local infections, F: Number of *antemortem* teeth lost. Dashed lines indicate 95% confidence intervals. Red lines indicate values for Dmanisi.

Fig. 14. Comparing the pattern of dentognathic pathologies in the Dmanisi specimens D2735/D211/D2600 and in matching triplets of modern HG specimens. Histograms represent frequency distributions of between-triplet dissimilarity (*D*), as measured between Dmanisi and modern HGs, and among modern HGs. Further explanations see text.

APPENDIX I

Class 1: Occlusal patterns and pathologies. Intra and inter-arch modifications resulting in suboptimal occlusal patterns.

- Features 1a to 1f: Tooth morphology and spacing. Presence/absence scoring: a) dental malformation; b) dental misorientation (tilt and/or rotation); c) crowding; d) spacing; e) ectopic tooth; f) dental mislocation (transposition, or incorrect location).
- Feature 1g: *In-vivo* tooth number. Scoring scheme: 0: normodontia [32 teeth]; -3: anodontia; -2: oligodontia; -1: hypodontia; 1: hyperdontia.
- Feature 1h: Occlusal pattern of the front dentition - Overbite and overjet. Scoring scheme: 0: normal/optimal; -4: negative-open; -3: negative; -2: positive-deep-open; -1: deep positive; 1: edge-to-edge; 2: cross bite; 3: open. In heavily worn dentitions, edge-to-edge bite was classified as a normal wear-related effect rather than a pathological feature (Kaifu et al. 2003).
- Feature 1i: Permanent M1 occlusal pattern, or the deviation from optimal molar relationship. Scoring scheme: 0: upper mesio-buccal cusp fits into lower anterior buccal groove (normal bite); -1: lower anterior buccal groove is distal to upper mesio-buccal cusp; 1: lower anterior buccal groove is mesial to upper mesio-buccal cusp; 2: cross bite – the transverse relation is misaligned; 3: open bite - the upper and lower M1s are not in occlusion.

Class 2: Non-carious lesions: Generalized and local dental lesions affecting the crown.

- Feature 2a and 2b: Hypoplasias and fluorosis. Scoring scheme: 0: absent; 1: <1/3 of the tooth crown affected; 2: 1/3-2/3 of the tooth crown affected; 3: >1/3 of the tooth crown affected. 2a) Linear enamel hypoplasia - a depressed horizontal furrow on the enamel around the circumference of the tooth crown (Hannibal and Guatelli-Steinberg 2005) and patchy enamel hypoplasia – hypomineralized areas are shaped as small pits (Clarkson and O'Mullane 1989); 2b) fluorosis – fluoride interaction with mineralizing tissues causing hypomineralisation, porosity and coloring (depending on the level of fluoride interaction the appearance may change) (Alvarez et al. 2009).
- Features 2c to 2f: Other non-carious lesions. Presence/absence scoring: 2c) wedge-shaped defect; 2d) erosive tooth wear; 2e) amelogenesis imperfecta; 2f) dentinogenesis imperfecta.

Class 3: Dentognathic trauma.

- Feature 3a: Enamel chipping. Scoring scheme: 0: absent; 1: small [<2mm]; 2: moderate [2-4]; 3: large [>4mm]. Every defect (even if on the same tooth) is taken into account.

- Feature 3b: Dental fracture where dentine is fractured together with enamel. Scoring scheme: 0: absent; 1: uncomplicated fracture [no pulp exposure]; 2: complicated fracture [pulp exposure]; 3 root fracture [root fracture only, cementum, dentine and pulp]; 4 crown-root fracture [enamel, dentine and root cementum; pulp may or may not be included].
- Features 3c to 3i: Mandibular fractures: 3c) symphyseal (presence/absence); 3d) dentoalveolar; 3e) lateral body; 3f) angle; 3g) ramus, condyle; 3h) ramus, coronoid process; 3i) ramus, condyle and coronoid process). Scoring scheme for features 3d to i: 0: absent; 1: single; 2: double and/or bilateral; 3: multiple.
- Features 3j to 3l: Maxillary fractures. Scoring scheme: 0: absent; 1: single; 2: double and/or bilateral; 3: multiple; 3k) present anterior; 3l) postcanine; 3m) maxillary tuber.
- Feature 3m: Maxillary fracture class after Le Fort (Le Fort 1901). Scoring scheme: 0: absent; 1: Le Fort 1; 2: Le Fort 2; 3: Le Fort 3 (Le Fort 1901).

Class 4: Reactive structures due to stress factors such as tooth wear, trauma, heavy masticatory loading, and others.

- Feature 4a: Hypercementosis. Scoring scheme: 0: absent; 1: mild [1/3 of the tooth roots affected in 2 to 4 teeth], 2: moderate [1/2 of the tooth root modification with more than 2 teeth affected]; 3: moderate to severe [>1/2 of the tooth root modification with >8 teeth affected; 4: severe [all teeth affected (to various degrees)].
- Feature 4b: Pulp chamber reduction. Scoring scheme: 0: absent; 1: reduction of the radicular pulp chamber region; 2: reduction of the radicular and coronal pulp chamber region; 3: pulp chamber reduced to small channel; 4: complete closure of the pulp chamber. Reduction of the pulp chamber in a single tooth is not considered as a general, age-related phenomenon. Four or more teeth must be involved.

Class 5: Dental caries.

- Features 5a to5 c: Location of dental caries. Presence/absence scoring: 5a) occlusal; 5b) approximal; 5c) cervical/root. Scoring scheme: 0: absent; 1: superficial with only enamel involved; 2: moderate with enamel and enamel- dentine junction (EDJ) destruction; 3: deep with enamel, EDJ and dentine involved; 4: deep with pulp exposure, involving enamel, EDJ, dentine and pulp chamber.

Class 6: Local infections, addressing inflammations and modification of the dental surrounding tissues.

- Feature 6a: Pathologic periodontal pocket. Scoring scheme: 0: absent, 1: mild, <1/3 of the interdental alveolar crest resorption; 2: moderate, 1/3-1/2 of the interdental alveolar crest resorption; 3: deep, more than 1/2 of the interdental alveolar crest resorption.

- Feature 6b: Inflammations of the periodontal tissues. Scoring scheme: 0: absent; 1: local periodontitis, 2: periapical granuloma/cyst; 3: periapical abscess; 4: osteomyelitis.

Class 7: Periodontal diseases.

- Feature 7a: dental plaque and calculus. Scoring scheme: 0: absent; 1: absence of calculus, but presence of black or brown plaque; 2; mild [1/4 of the tooth surface covered with calculus]; 3: moderate [<1/2 of the tooth surface covered with calculus; 4: severe [>1/2 of the tooth surface covered with calculus].
- Feature 7b: Root furcation involvement. Scoring scheme: 0: absent; 1: <3mm; 2: >3mm; 3: fully probed (furcation can be trespassed through in bucco-lingual direction) 4: root is stripped to the halfway or more.
- Feature 7c: Alveolar bone structural changes due to periodontal disease. Scoring scheme: 0: absent; 1: mild, alveolar margins blunt and flat-topped; 2: moderate, alveolar margins rounded and porous, 2-4 mm depth between the tooth and alveolus; 3: severe, alveolar margins ragged and porous, irregular trough of funnel, >5mm depth between the tooth and the alveolus.

Class 8: *Antemortem* tooth loss: number of teeth lost, and post-loss condition of the residual alveolar ridge.

- Feature 8a: Number of lost teeth. Scoring scheme: 0: none; 1: <5 teeth lost; 2: 5 to 9 teeth lost; 3: 10 to 14 teeth lost; 4: 14 or more teeth lost.
- Feature 8b: Resorption level of the residual alveolar ridge. Scoring scheme: 0: none; 1: smooth alveolus, but observable socket, 2: very smooth alveolus no observable socket; 3: complete resorption of the alveolar bone. In edentulous specimens such as the D3900 mandible alveolar bone resorption was also measured as the distance from the alveolar plane to the deepest point of the residual ridge (Fig. 12).

Class 9: Temporomandibular joint (TMJ) disorder.

- Feature 9a: TMJ degenerative processes. Scoring scheme: 0: absent: 1: mild, 2: moderate; 3: moderate but strong lipping (bony overgrowth at the edges), 4: severe.

The classification and scoring scheme was used to record DP in each specimen of the sample. Additionally, per-specimen summary scores were established for dentognathic pathologic features with multiple locations. For example, if occlusal dental caries is present in two teeth, and scored 1 (superficial) and 2 (moderate), respectively, the summary score for caries is 3.

Name	N	Geographical locality	Geographical locality	Coordinates	Climatic zone	Data*
D2735		Caucasus	Dmanisi	41.2010 N, 44.2038 E	Temperate	CT, µCT, SR-µCT
D2678 LRC, D2723 LLC, D2854 LR I2, D3698 LLI2		Caucasus	Dmanisi	41.2010 N, 44.2038 E	Temperate	µCT
D211		Caucasus	Dmanisi	41.2010 N, 44.2038 E	Temperate	CT
D2600		Caucasus	Dmanisi	41.2010 N, 44.2038 E	Temperate	CT, SR-µCT
D3900		Caucasus	Dmanisi	41.2010 N, 44.2038 E	Temperate	CT
Australian	26	West Australia	Dampier (9)	~20.66 S, 116.71 E	Hot	CT
			Fremantle (1)	~32.05 S, 115.74 E	Temperate	
			Baiong (1)	~22.60 S, 114.39 E	Hot and Dry	
		Queensland	Croydon (2),	~18.12 S, 142.14 E	Hot and Dry	
			Maranoa (1)	~26.42 S, 145.52 E	Hot and Dry	
			between Seven and Batavia Rivers (2)	~12.13 S, 142.06 E	Tropical	
		South Australia	Adelaide (4)	~34.92 S, 138.60 E	Temperate	
		New South Wales	Berida (1)	~31.64 S, 148.44 E	Temperate/Grassland	
		Victoria	(1)	~37.18 S, 144.64 E	Temperate	
		Unknown	Woolangala tribe (1)	?	?	
		Torres	Hammond island (2)	~20.05 S, 148.88 E	Tropical	
			Mabuiag island (1)	~9.95 S, 142.18E	Tropical	
Greenlander	47	AIM collection:				CT
		South-East	Ammassalik (15)	~65.61 N, 37.63 W	Arctic	
		Unknown	(1)	?	?	
		Greenland Museum collection:				
		South-East	Ammassalik (3);	~65.61 N, 37.63 W	Arctic	
			Akorninap Kangerlua (2);	~~64.55 N, 33.94 W	Arctic	
		South-West	Inussuk (2);	~64.16 N, 51.72 W	Arctic	
			Kangaamiut (2);	~65.82 N, 53.34 W	Arctic	
			Uummannalik (3);	~60.61 N, 46.05 W	Arctic	
		North-East	ClaveringØ (4);	~ 74.22 N, 21.14 W	Arctic	
			Suess Land (2);	~~77.93 N, 24.10 W	Arctic	
			Scoresby Land (1);	~70.39 N, 25.31 W	Arctic	
			Dove Bugt (1);	~76.50 N, 19.11 W	Arctic	
		Noth-West	Upernavik (3);	~72.78 N, 56.13 W	Arctic	
			Anap Nunaa (6);	~69.93 N, 50.54 W	Arctic	
			Nuussuaq (2).	~74.11 N, 57.06 W	Arctic	

				Unknown (1)	~~80.65 N, 61.56 W	Arctic		
		East		Nordfjord (1)	~66.64 N, 37.61 W	Arctic		

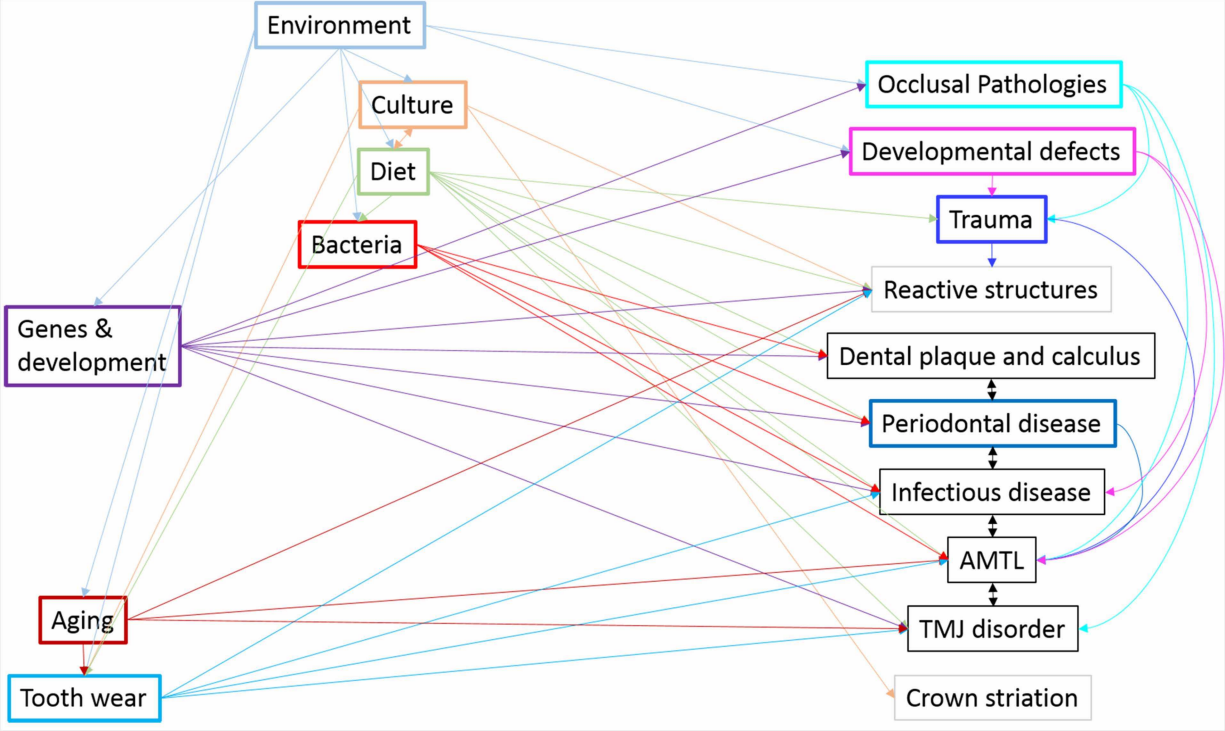
Table 1
Sample structure
*data: CT: measurements taken and analyzed from CT data of original specimens (these specimens were used in all analyses), μCT measurements from microCT data of the original specimens, SR-μCT measurements from synchrotron tomography data of the original specimens.

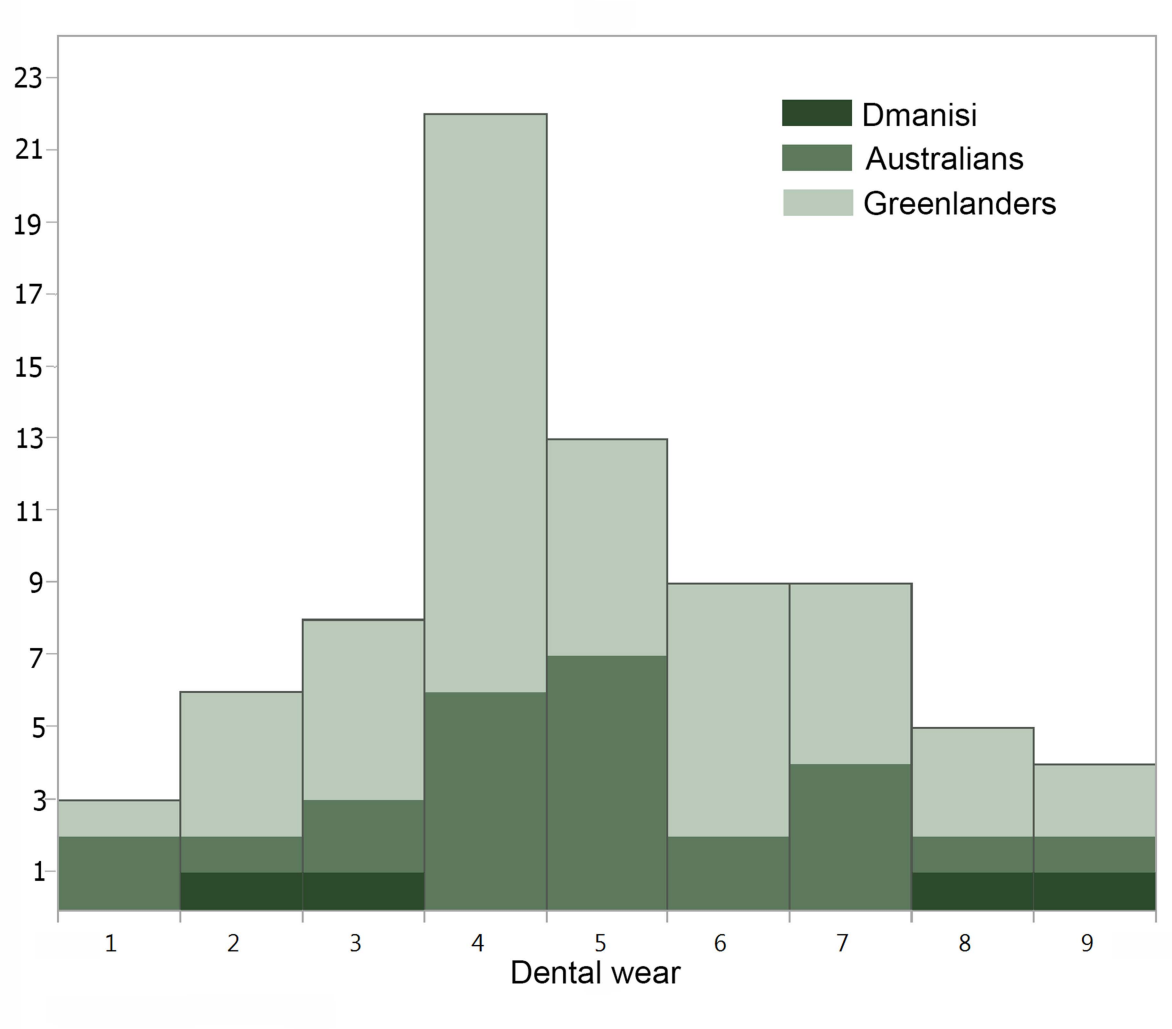
Abbreviation	Full name
AMTL	*Ante mortem* tooth loss
AP	Antero-posterior
C	Canine
CEJ	Cemento-enamel junction
CT	Computed tomography
DP	Dentognathic pathologies
EDJ	enamel-dentine junction
EH (LEH)	Enamel hypoplasia (Linear enamel hypoplasia)
HG	Hunter-gatherer
I	Incisor
JEOL	Japan Electron Optics Laboratory
L	Left
M	Molar
ML	Medio-lateral
µCT	Micro computed tomography
N	Number
P	Premolar
R	Right
SEM	Scanning electron microscope
SR-µCT	Synchrotron tomography
TMJ	Temporomandibular joint

Table 2
Abbreviations used in the study.

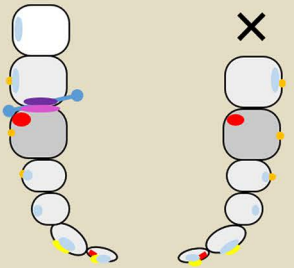
Class	1a	1b	1c	1f	1g	1g	1i	2a	3a	3b	4a	4b	5a	5b	5c	5	6a	6b	7a	7b	7c	8a	8b	9a	
Feature	malformation	rotation and tilt	crowding	incorrect eruption	number of teeth/hypodontia	number of teeth/hyperdontia	M1/M1 relation/distal shift	enamel hypoplasia	enamel chipping	trauma dental fractures	hypercementosis	pulp chamber reduction	occlusal caries	approximal caries	cervical caries	severity of caries expression	pathologic periodontal pocket	local infections	dental plaque and calculus	furcation visibility	periodontal disease	N AMTL	Alv. bone remodel. AMTL	TMJ disorder	Total N of individuals
A																									
Dmanisi	0	1	1	0	1	0	0	2	3	1	1	1	0	1	0	1	1	2	3	3	3	2	2	1	4
Australia	0	1	2	2	1	1	2	10	6	2	3	11	4	5	1	9	11	10	18	14	18	6	6	3	26
Greenland	1	8	2	0	4	0	4	19	29	5	5	29	0	0	0	0	21	24	37	38	37	17	17	2	49
B																									
Dmanisi	0	0.25	0.25	0.00	0.25	0.00	0.00	0.50	0.75	0.25	0.25	0.25	0.00	0.25	0.00	0.25	0.25	0.50	0.75	0.75	0.75	0.50	0.50	0.25	
Australia	0	0.04	0.08	0.08	0.04	0.04	0.08	0.38	0.23	0.08	0.12	0.42	0.15	0.19	0.04	0.35	0.42	0.38	0.69	0.54	0.69	0.23	0.23	0.12	
Greenland	0	0.16	0.04	0.00	0.08	0.00	0.08	0.39	0.59	0.10	0.10	0.59	0.00	0.00	0.00	0.00	0.43	0.49	0.76	0.78	0.76	0.35	0.35	0.04	

Table 3
Prevalence of pathology features in Dmanisi, Australian, and Greenlander samples. A: absolute numbers of affected individuals/teeth. B: normalized data. Color code: white – absent; light to dark grey – low to high prevalence.

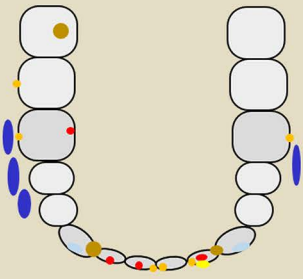

Environment
Culture
Diet
Bacteria
Genes & development
Aging
Tooth wear
Occlusal Pathologies
Developmental defects
Trauma
Reactive structures
Dental plaque and calculus
Periodontal disease
Infectious disease
AMTL
TMJ disorder
Crown striation


Dmanisi
Australians
Greenlanders
23
21
19
17
15
13
11
9
7
5
3
1
1
2
3
4
5
6
7
8
9
Dental wear

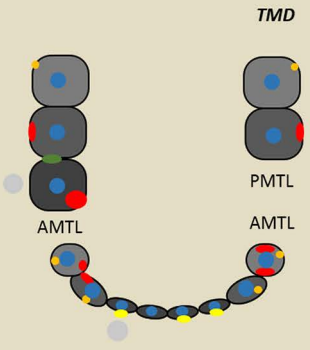
2cm



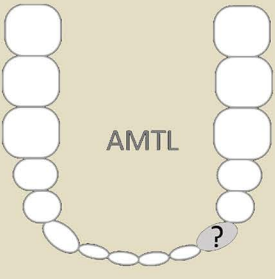
D2735



D211



D2600



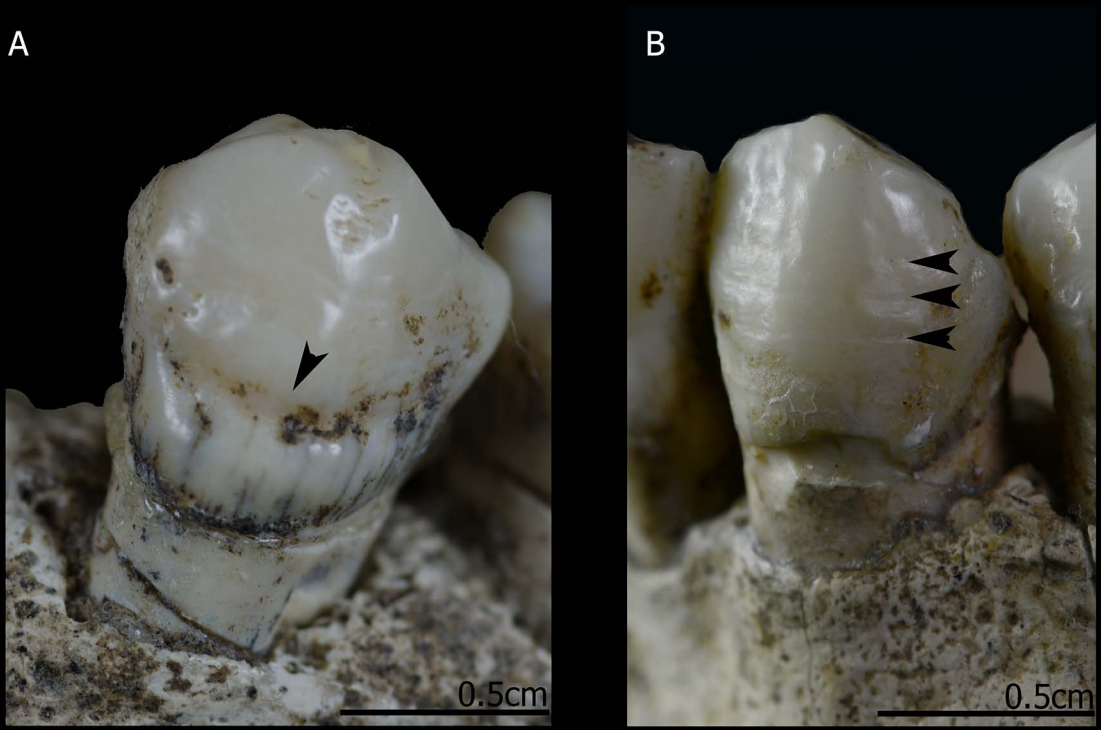
D3900

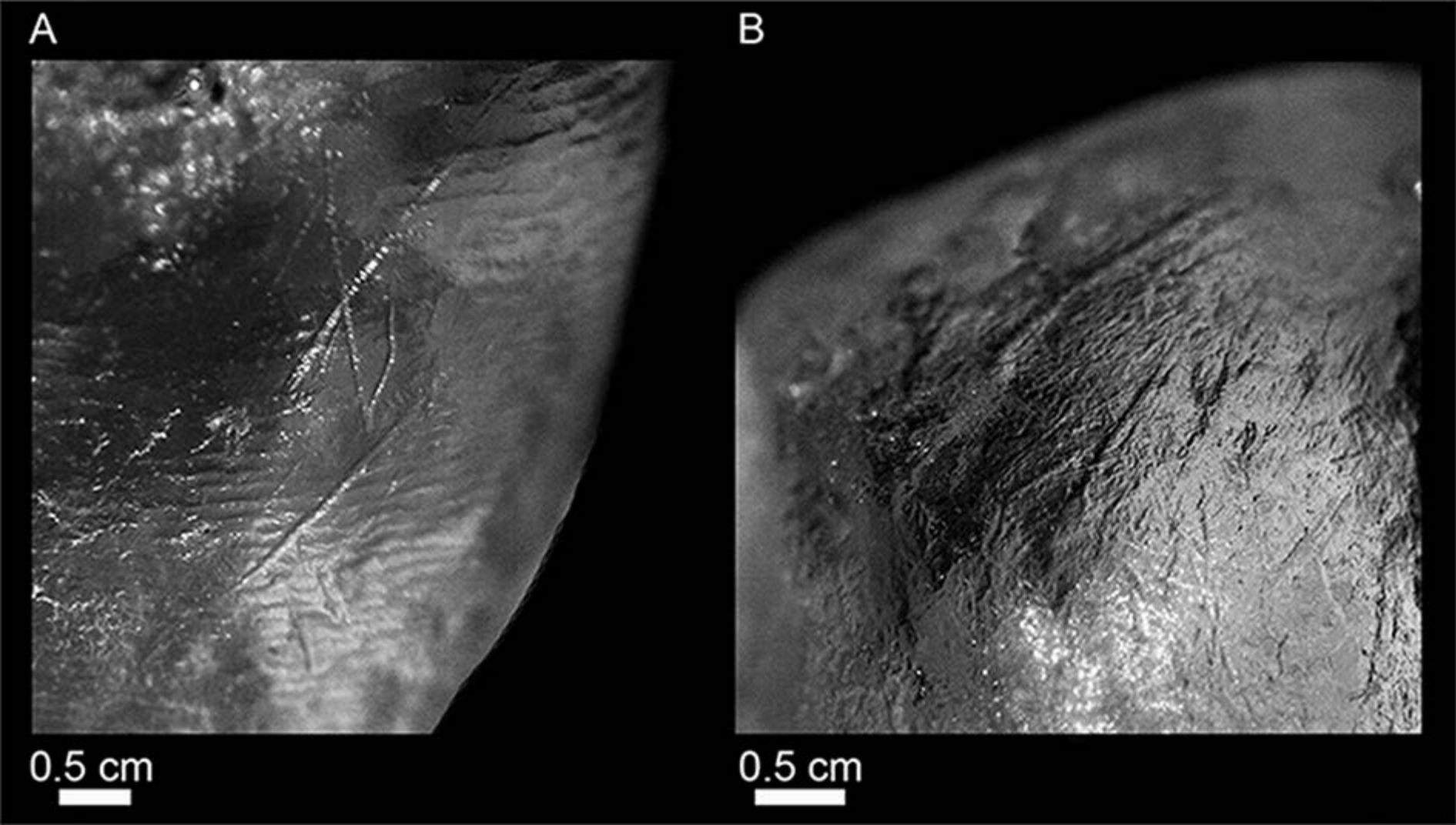
- ✖ Agenesis
- Enamel hypoplasia
- Enamel scratches
- Enamel chipping/crown fracture
- Toothpick lesion
- Pathologic periodontal pocket
- Marginal periodontitis
- Calculus
- Crowding
- Periodontal disease
- Hypercementosis
- Caries
- Periapical cyst

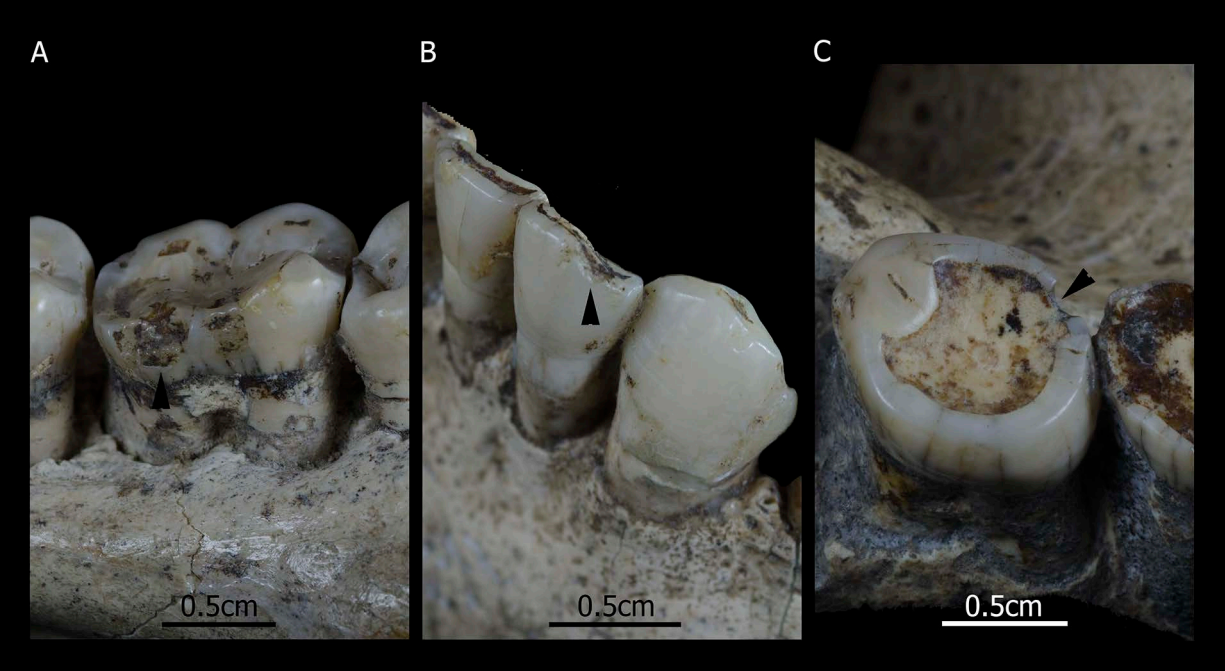
AMTL *Ante mortem* tooth loss

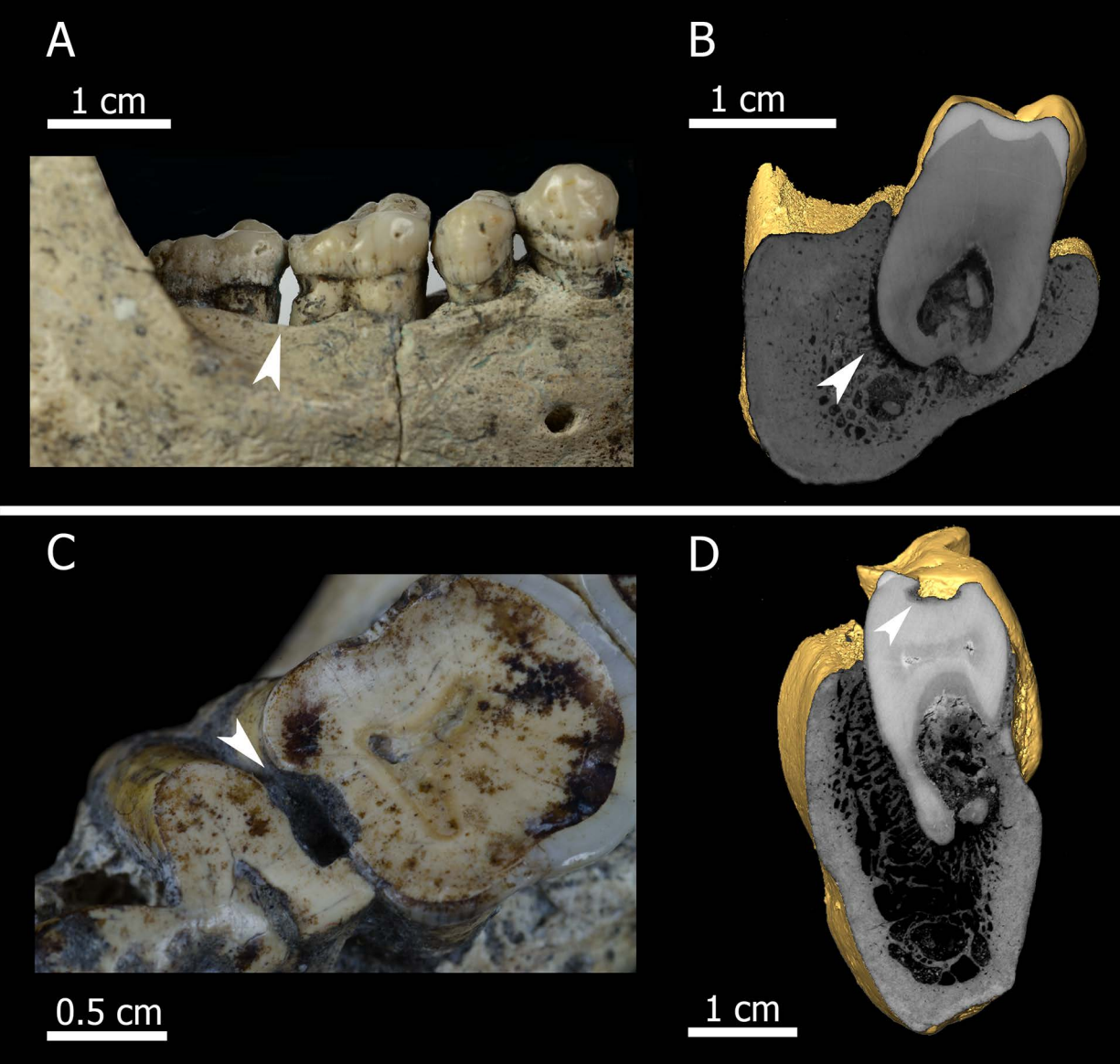
PMTL *Post mortem* tooth loss

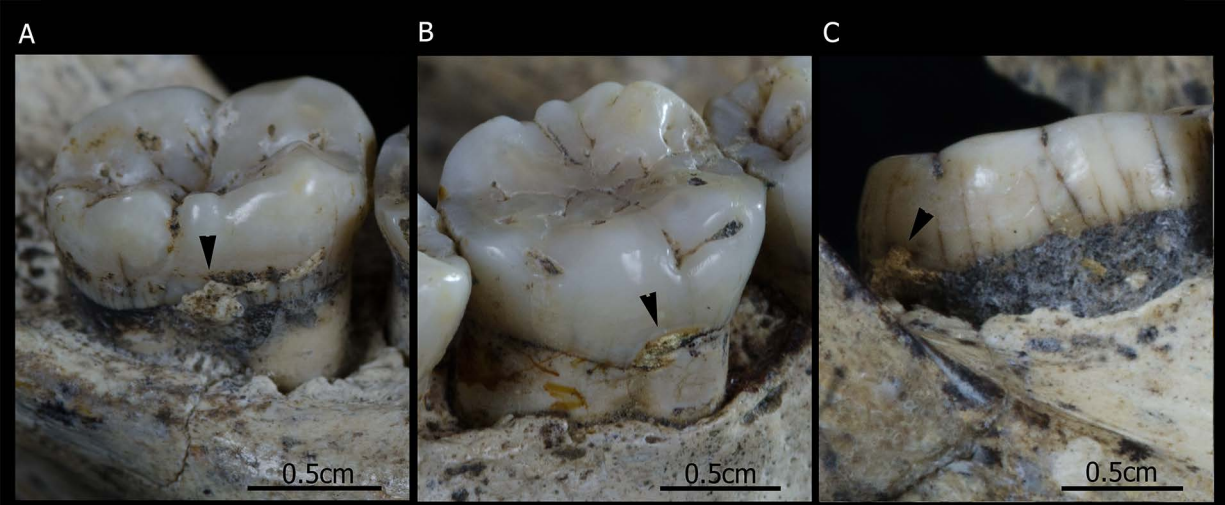
TMD Temporomandibular joint disorder

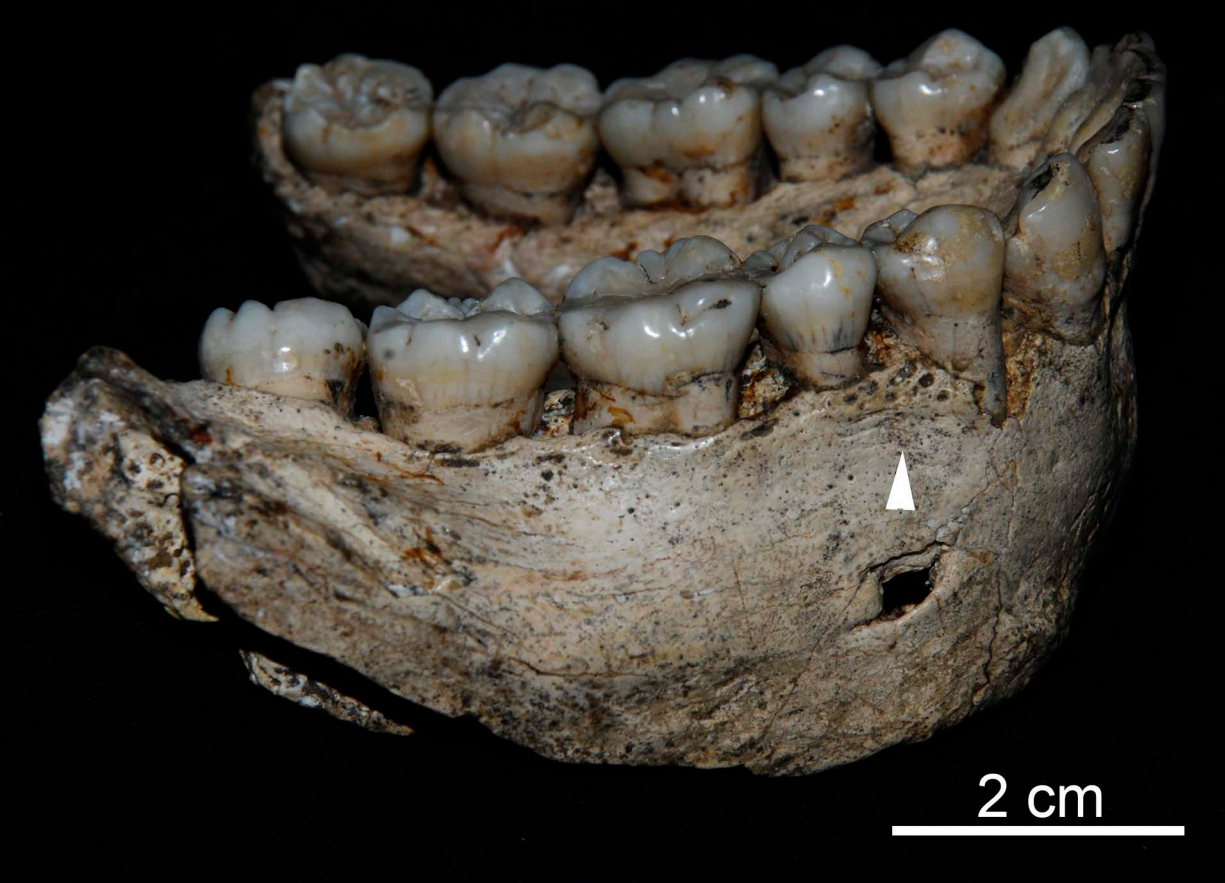

A
0.5cm
B
0.5cm

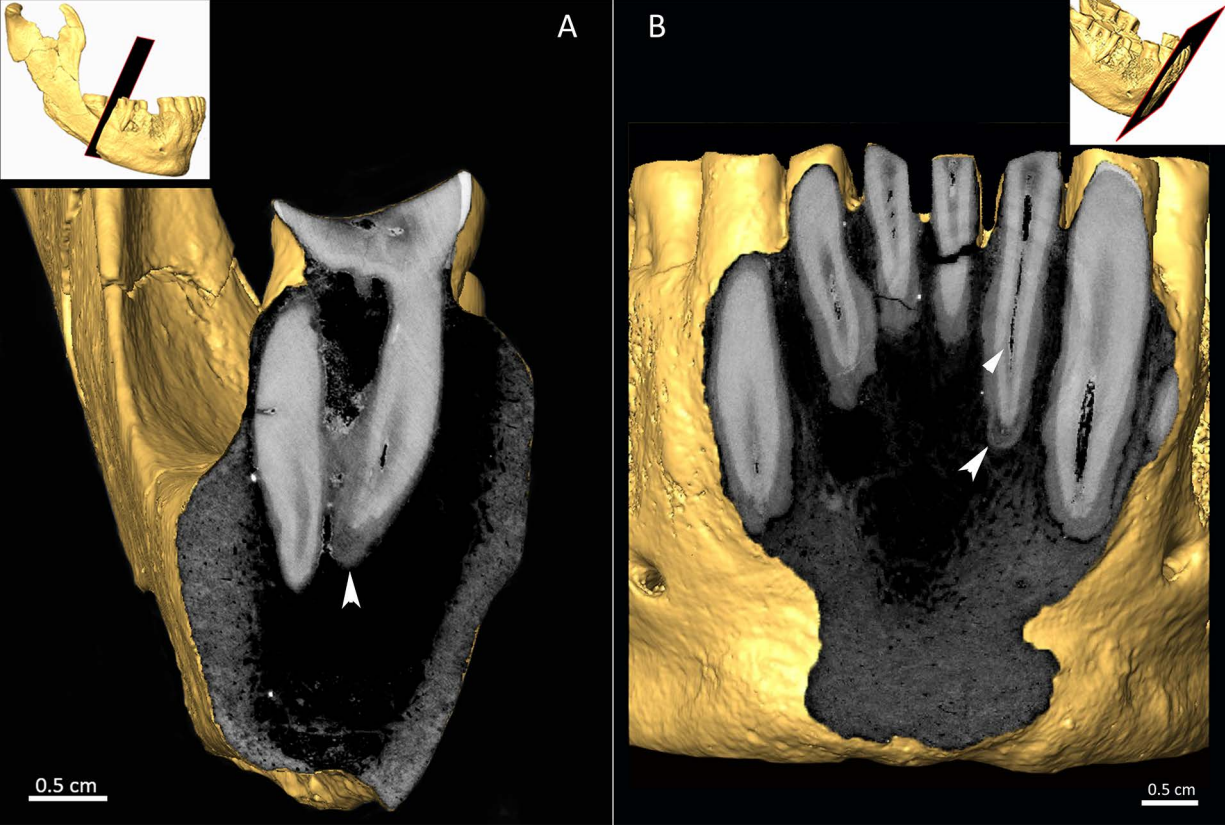

A
0.5 cm
B
0.5 cm

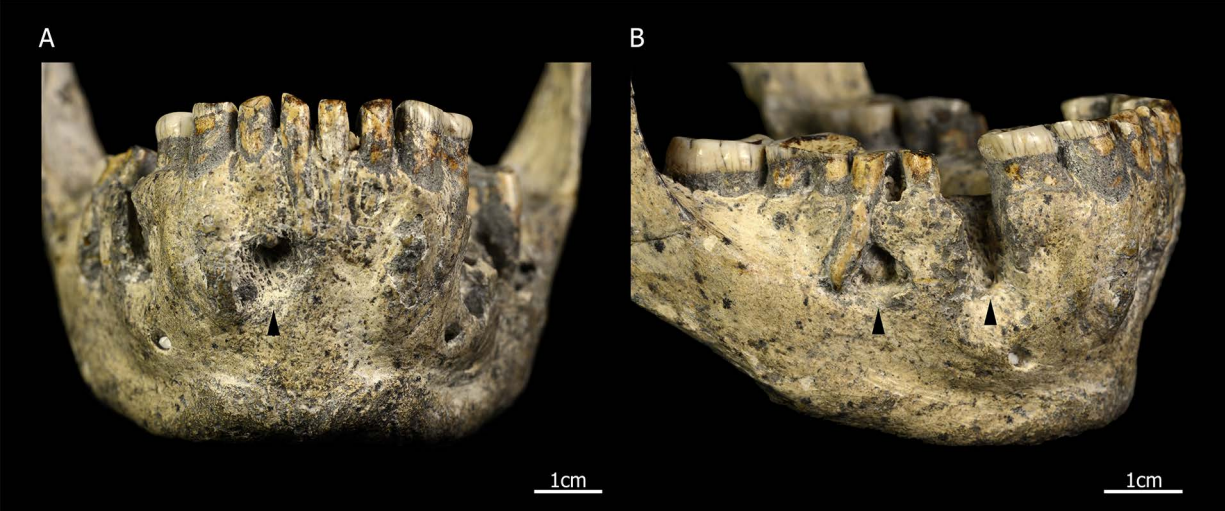

A
B
C
0.5cm
0.5cm
0.5cm

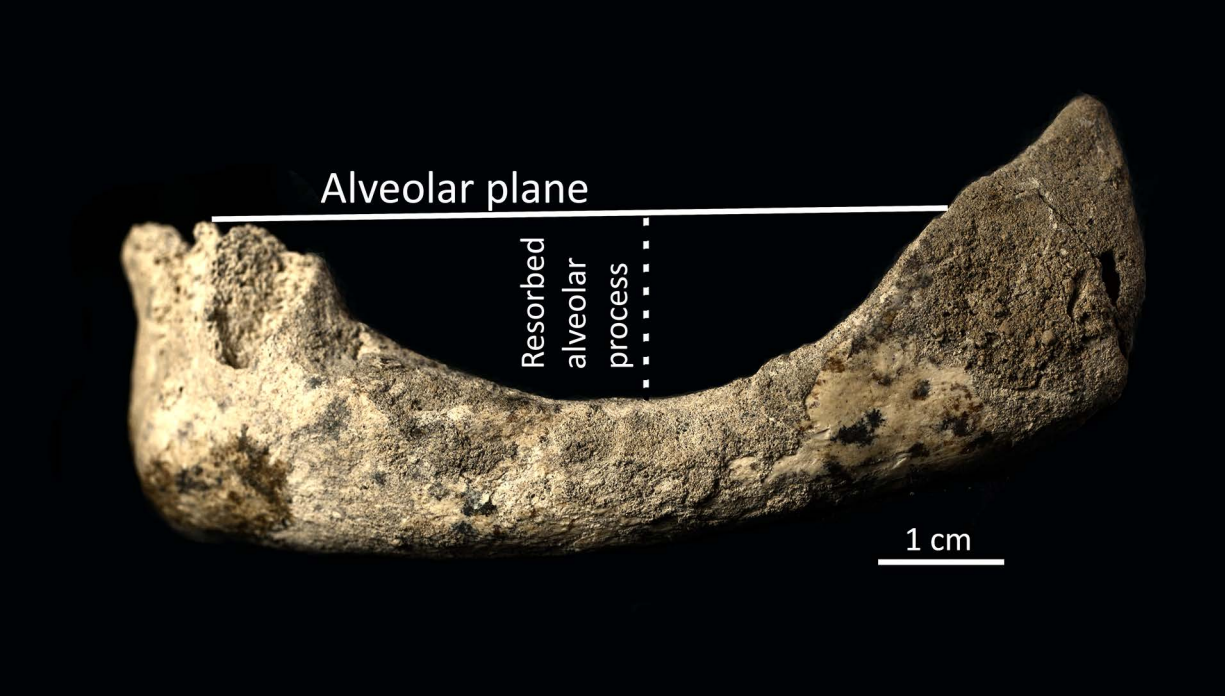

A
1 cm
B
1 cm
C
0.5 cm
D
1 cm

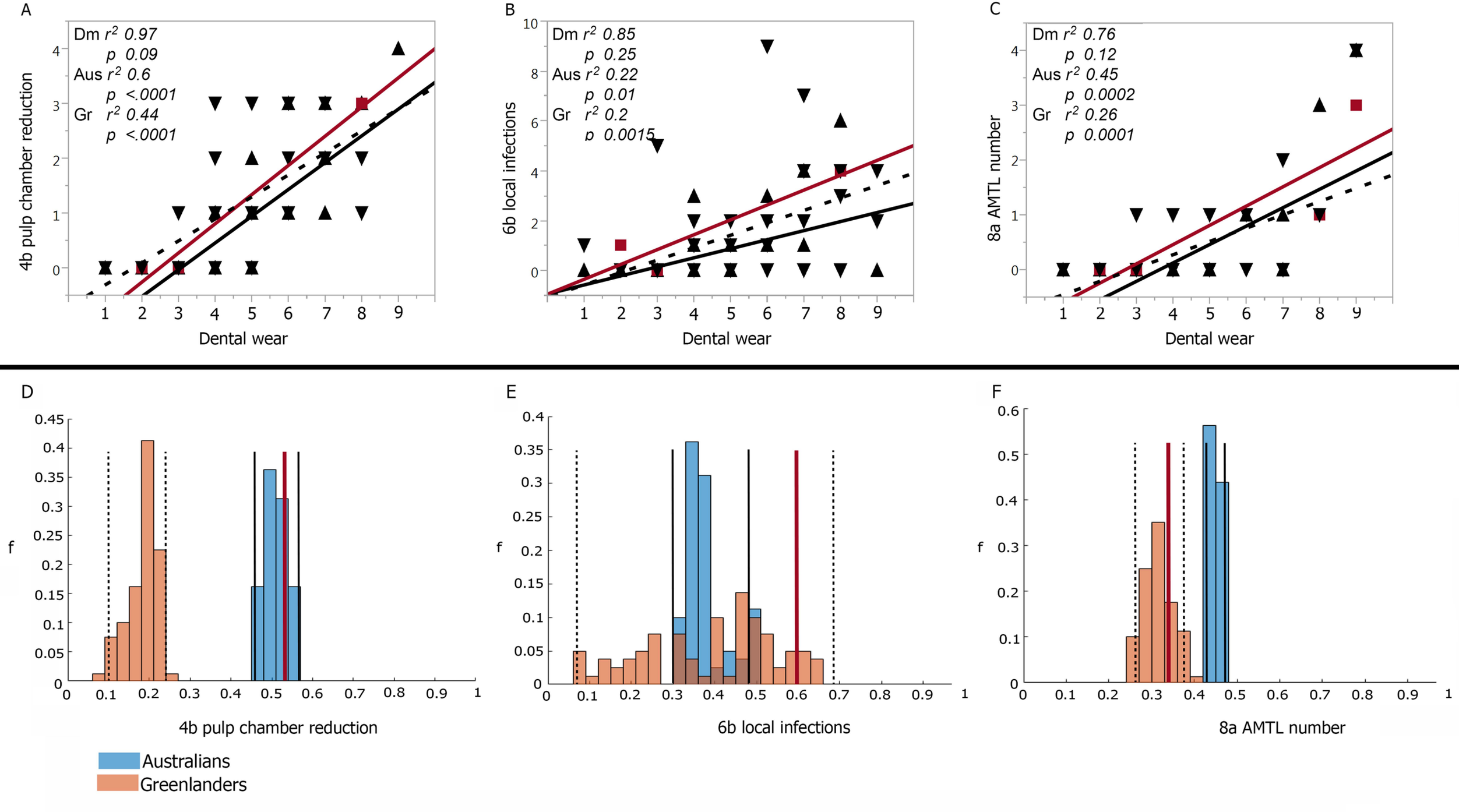

A
0.5cm
B
0.5cm
C
0.5cm

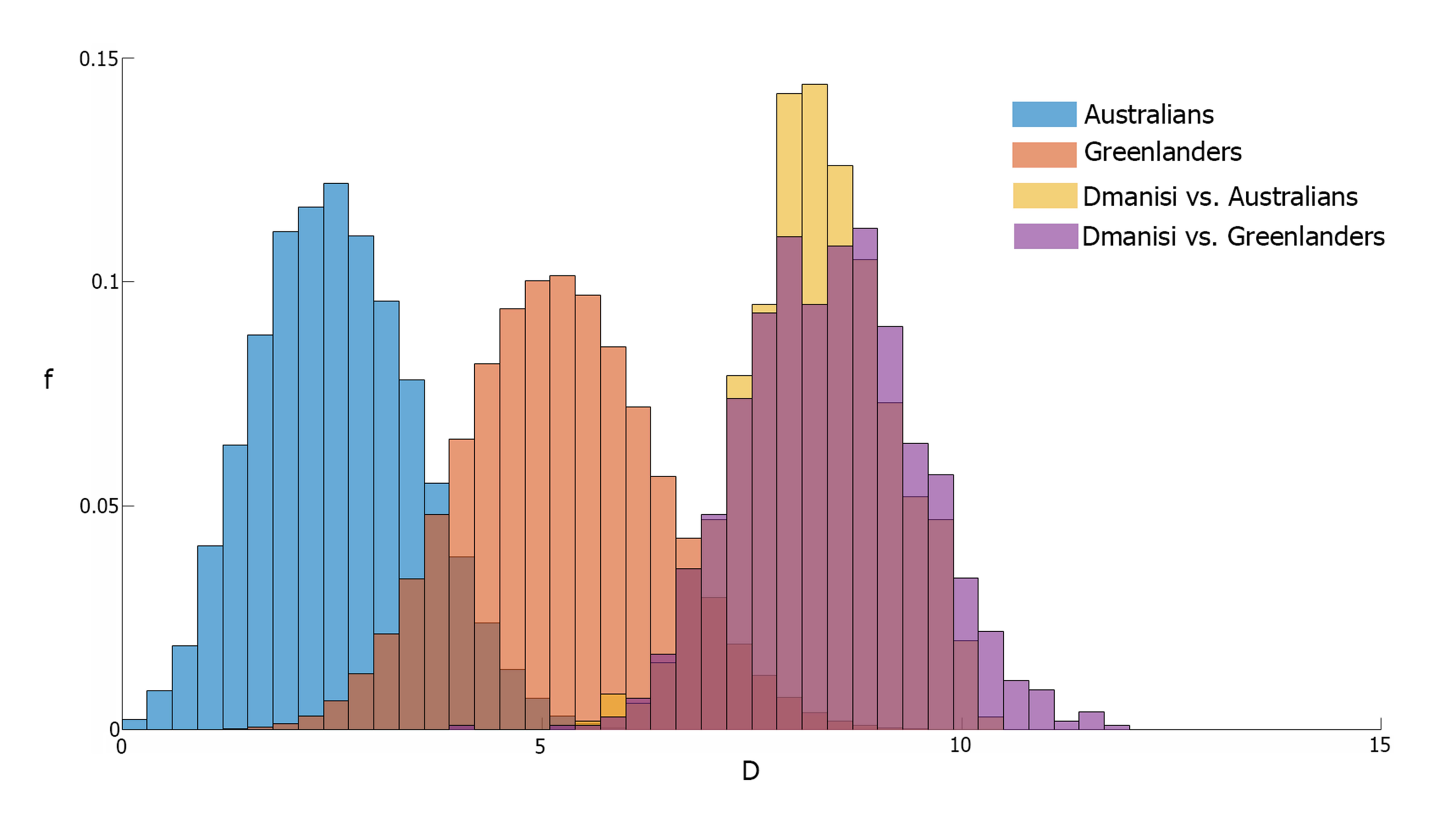

2 cm


A
B
0.5 cm
0.5 cm


A
1cm
B
1cm


Alveolar plane
Resorbed alveolar process
1 cm


A
Dm r^2 0.97
p 0.09
Aus r^2 0.6
p <.0001
Gr r^2 0.44
p <.0001
4b pulp chamber reduction
Dental wear
B
Dm r^2 0.85
p 0.25
Aus r^2 0.22
p 0.01
Gr r^2 0.2
p 0.0015
6b local infections
Dental wear
C
Dm r^2 0.76
p 0.12
Aus r^2 0.45
p 0.0002
Gr r^2 0.26
p 0.0001
8a AMTL number
Dental wear
D
f
4b pulp chamber reduction
E
f
6b local infections
F
f
8a AMTL number
Australians
Greenlanders


0.15
0.1
0.05
0
f
0
5
10
15
D
Australians
Greenlanders
Dmanisi vs. Australians
Dmanisi vs. Greenlanders

SI Table 1

Dentognathic pathologies coding for permanent dentition per jaw

Class	Feature	description	-4	-3	-2	-1	0	1	2	3	4
1a	**occlusal pattern**	*malformation*					absent	present			
1b	**occlusal pattern**	*rotation/tilt*					absent	present			
1c	**occlusal pattern**	*crowding*					absent	present			
1d	**occlusal pattern**	*spacing*					absent	present			
1e	**occlusal pattern**	*ectopic tooth*					absent	present			
1f	**occlusal pattern**	*transposition, or mislocation*					absent	present			
1g	**occlusal pattern**	*number of teeth*		anodontia	oligodontia	hypodontia	32 teeth	hyperdontia			
1h	**occlusal pattern**	*overbite and overjet*	negative -open	underbite /negative	positive-deep-open	deep	optimal, 1/3, < 1/3, edge-to edge|according to wear rate	edge-to-edge	cross	open	
1i	**occlusal pattern**	*mandibular relation towards maxilla, M1 occlusal pattern*				distal	Upper MB-cusp fits into Lower V-groove	mesial	cross	open	
2a	**non-carious lesions**	*linear or patchy (pits) enamel hypoplasia*					absent	< 1/3	1/3-2/3	>1/3	
2b	**non-carious lesions**	*fluorosis*					absent	< 1/3	1/3-2/3	>1/3	
2c	**non-carious lesions**	*wedge shaped defect*					absent	present			
2d	**non-carious lesions**	*erosive tooth wear*					absent	present			
2e	**non-carious lesions**	*amelogenesis imperfecta general*					absent	present			
2f	**non-carious lesions**	*dentinogenesis imperfecta*					absent	present			
3a	**trauma**	*enamel chipping*					absent	small <2mm	moderate 2-4mm	large >4mm	
3b	**trauma**	*fractured tooth*					absent	uncomplicated fracture, no pulp exposure	complicated fracture, pulp exposure	root fracture, cementum, dentine and pulp	crown-root fracture, enamel, dentine and root cementum; pulp may or may not be included
3c	**trauma**	*fractured mandible/location/ symphyseal(anterior)*					absent	present			
3d	**trauma**	*fractured mandible/location/dentoalveolar*					absent	present			
3e	**trauma**	*fractured mandible/location/lateral body*					absent	present			
3f	**trauma**	*fractured mandible/location/angle*					absent	present			
3g	**trauma**	*fractured mandible/location/ ramus condyle*					absent	present			
3h	**trauma**	*fractured mandible/location/ ramus coronoid*					absent	present			
3i	**trauma**	*fractured mandible/location/ ramus condyle and coronoid*					absent	present			
3j	**trauma**	*fractured maxillary/location/ anterior*					absent	present			
3k	**trauma**	*fractured maxilla/location/ postcanine*					absent	present			
3l	**trauma**	*fractured maxilla/location/ tuber*					absent	present			
3d-i/scor	**trauma**	*fractured mandible*					absent	single	double (and bilateral)	multiple	
3j-l/scori	**trauma**	*fractured maxilla*					absent	single	double (and bilateral)	multiple	
3m	**trauma**	*fractured maxilla/class*					absent	Le Fort 1	Le Fort 2	Le Fort 3	
4a	**hypercementosis**	*cementum remodeling*					absent	mild: 1/3 of the tooth modification. 2 - 4 teeth affected	moderate: 1/2 of the tooth modification. more than 2 teeth affected	moderate to severe: >1/2 of the tooth modification. more than 8 teeth affected	severe: all teeth are modified
4b	**reactive structures**	*pulp chamber reduction*					absent	reduction of the radicular pulp	reduction of the radicular and coronal pulp	presence of the small gap in the radicular pulp	nearly complete closure of the pulp chamber
5a	**caries**	*location/occlusal*					absent	present			
5b	**caries**	*location/approximal*					absent	present			
5c	**caries**	*location/cervical,root*					absent	present			
5a-c/scor	**caries**	*severety*					absent	superficial (E)	moderate (E, EDJ)	deep (E, EDJ, D - without pulp exposure)	deep with pulp exposure (E, EDJ, D,P)
6a	**local infections**	*pathological pocket, deep gap of the gingival sulcus*					absent	mild: smallar than 1/3	moderate: 1/3 to 1/2	deep: more than 1/2	
6b	**local infections**	*inflamation of surrounding tissues*					absent	local periodontitis	periapical granuloma/cyst	periapical abscess	osteomielitis
7a	**dental plaque and calculus**	*plaque and calculus*					absent	absence of calculus (black or brown plaque present)	mild: <1/4th of thoos surface covered with calculus	moderate: <1/2 of tooth covered with calculus	severe: >1/2 of tooth covered with calculus
7b	**periodontal disease**	*furcation involvment*					absent	<3mm	>3mm	fully probed furcation	root stripped to the halfway or more
7c	**periodontal disease**	*expression*					absent	mild: alveolar margins blunt and flat-topped	moderate: alveolar margins rounded and porous, 2-4 mm depth between tooth and alveolus	severe: alveolar margins ragged and porous, irregular trough of funnel >5mm depth between tooth and alveolus	
8a	***ante-mortem* tooth loss**	*number of lost teeth (per jaw)*					absent	<5 teeth lost	5 to 9 teeth lost	10 to 14 teeth lost	>14 teeth lost
8b	***ante-mortem* tooth loss**	*residual ridge resorption level*					absent	smooth alveolus, but observable socket	very smooth alveolus, no observable socket	complete resorption of alveolar bone	
9a	**TMJ disorder**	*TMJ degenerative processes*					absent	mild	moderate	moderata but strong lipping	severe
								Le Fort 1: horizontal maxillary fracture, separating the teeth from the upper face fracture line passes through the alveolar ridge, lateral nose and inferior wall of maxillary sinus	Le Fort 2: pyramidal fracture, with the teeth at the pyramid base, and nasofrontal suture at its apex fracture arch passes through posterior alveolar ridge, lateral walls of maxillary sinuses, inferior orbital rim and nasal bones	Le Fort 3: craniofacial disjunction fracture line passes through nasofrontal suture, maxillo-frontal suture, orbital wall and zygomatic arch	